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ASCORBIC ACID IN THE MILK OF MELBOURNE WOMEN.

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As a sequel to the work on the mineral content of breast milk,[1] an investigation was carried out on the ascorbic acid content of maternal milk in the Melbourne area.

Literature.

In man, according to Boyle,[2] normal ascorbic acid nutrition is essential for the maintenance of the tissues by which the teeth are attached to the jaws. Loosening of the teeth in ascorbic acid deficiency is the result of atrophy of such tissues. This author also considered such suboptimal ascorbic acid nutrition to be an underlying factor in the production of systemic pyorrhœa. Moreover, in infants and children receiving enough vitamin *C* for protection against scurvy, there may still develop a condition of latent or subacute scurvy accompanied by more or less severe injury to the teeth (Smith[3]). It thus becomes apparent that larger quantities of vitamin *C* are required for good nutrition than for the prevention of scurvy. For this reason, and in view of the widespread occurrence of dental caries in this country, there is an urgent necessity to safeguard the proper vitamin *C* nutrition of infants and children.

It has been proved beyond any doubt that artificially fed infants need a vitamin *C* supplement as early as the first week of life, owing to the low content of this vitamin in cow's milk compared with that in human milk. The practice of giving even breast-fed children orange juice after the first six months has also been adopted in this country. The question arises, however, whether in all cases there is an adequate vitamin *C* intake during the first six months of breast feeding.

The possibility that the infant has the ability of synthesizing vitamin *C* up to its tenth month of life, as suggested by Rohmer and Bezssonoff[4] and by Stoerr,[5][6] has been denied by Wachholder,[7] by Ferdinand,[8] by Muller,[9] by Neuweiler[10] and others, who express the opinion that although after birth the infant possesses a store of vitamin *C* which enables it to cover the deficit for some time, its only source of intake is breast milk.

According to Elmby *et alii*,[11] Baumann[12] and others, the ascorbic acid content of milk follows closely that of blood, which in turn is completely dependent on the diet of the nursing mother (Baumann and Rappolt,[13] Gedd and Kjelberg[14]).

Numerous experiments have been carried out to raise the ascorbic acid level in milk by means of a high vitamin *C* intake, either in the food or by the administration of the synthetic vitamin. The general belief is that the lactating mother has no special storage capacity for vitamin *C* once the saturation point has been reached (Neuweiler[15] Chu *et alii*,[16] Baumann[12]), although during lactation the body appears to store vitamin *C* for longer periods than usual (Baumann and Rappolt[13]). To quote from Baumann:[12]

> There is no lower threshold for secretion from blood into milk and it goes on until the maternal organism is greatly depleted, but there is an upper limit of about 8 mg. per cent. in milk. There is no upper limit for secretion into urine, but there is a lower limit about 1·1·4 mg. per cent. in blood.

However, with regard to the question of the ascorbic acid level in milk in the case of a deficiency in the diet, Laurin[17] and Widenbauer and Rappolt (see Laurin[17]) think that the amount available can if necessary be supplied to the child by means of breast milk, even at the cost of the mother's own need of this vitamin. Gaehtgens

and Werner,[18] on the other hand, express the opinion that when the daily requirements are not covered the mother's organism becomes depleted during gestation, but during lactation the infant is the sufferer, since the ascorbic acid content of the milk falls and the mother is thereby protected.

In view of this controversy it seems to be advisable to supplement the possible deficiency in milk by giving orange juice or other easily assimilated source of vitamin *C* direct to the infant rather than to the mother. However, as Smith[23] recommends, no harm is done if the intake of the nursing mother is doubled during the period of considerable drain on her resources.

Daily Requirements of Mother and Infant.

The Technical Committee on Nutrition of the League of Nations[19] has estimated the vitamin *C* requirements of artificially fed infants to be five to fifteen milligrammes per day. The majority of investigators, however, taking the average vitamin *C* intake of a breast-fed infant nursed by a mother receiving a satisfactory diet, consider this amount inadequate. Young[21] and Hess and Benjamin[20] recommend the daily allowance of 7·5 to 20 milligrammes to prevent avitaminosis, whilst Wachholder[7] considers 10 to 15 milligrammes per day as an absolute minimum. Smith,[2] summarizing the results of several investigators, estimates the daily requirements of an infant as five to eight milligrammes of ascorbic acid per kilogram of body weight, based on a daily intake during the first few months of 500 to 1,000 mils of milk with an ascorbic acid content within the range of four to seven milligrammes *per centum*. A concentration below four milligrammes *per centum* is regarded by this author and others as an indication of vitamin *C* deficiency in the tissues of lactating women.

Table I shows the respective daily intakes of mother and infant and the percentage of ascorbic acid in milk as given by different authors.

TABLE I.

Author.	Daily Intake of Ascorbic Acid.		Ascorbic Acid in Milk. (Milligrammes *per Centum*.)
	Mother.	Infant.	
Widenbauer and Kuhner	80 to 100 milligrammes	40 to 50 milligrammes	—
De Haas and Meulemans[22]	Adequate	40 milligrammes	4 to 5
Toverud[12]	100 milligrammes	25 to 30 milligrammes	6 to 7
Baumann[11]	50 milligrammes	8 to 14 milligrammes	—
Chu, Woo and Sung[13] ..	82 milligrammes	Adequate	—
Ingalls *et alii*[14]	—	28 milligrammes	4 to 5
Ingalls[15]	—	20 to 40 milligrammes	4
Selleg and King[16] ..	—	40 to 50 milligrammes	6 to 8
Bradstrup[17]	Adequate	3 to 8 milligrammes per kilogram of body weight	—
Bessey[25]	Adequate	20 to 40 milligrammes	—
Baumann[11]	70 milligrammes	—	4

Materials and Methods.

The investigation was carried out during the spring and summer of 1942-1943. The samples were taken at the Women's Hospital, Melbourne, and at several baby health centres and homes for mothers and babies. They represented mostly the lower and middle income groups.

The source of material and the method of collection of milk samples were the same as in the previous work.[1] Milk was collected in small, brown, glass-stoppered bottles, transferred to a black box and immediately taken to the laboratory. The estimation was carried out not later than two hours after collection.[1]

Tillman's method of estimation, as modified by Bessey,[25] was used, but as the end-point presented considerable difficulties, Kuhn's[24] back titration with Mohr's salt solution was applied and gave satisfactory results.[2]

Reagents.

The ascorbic acid solution was prepared as follows: 0·002 to 0·004 gramme was made up to 100 millilitres with 3% metaphosphoric acid solution.

The 2-6-dichlorophenol-indophenol was made up as follows: 0·01 gramme was dissolved in hot water. After cooling, 10 millilitres of phosphate buffer solution at pH 6·8 were added and the total volume was made up to 100 millilitres. The buffer solution consisted of 15·45 millilitres of 0·2 molar sodium phosphate solution and 4·55 millilitres of 0·1 molar citric acid solution kept over chloroform.

Mohr's stock solution was prepared in the following way: 0·1 gramme of ammonium ferrous sulphate (C.P.) was dissolved in water with two millilitres of concentrated sulphuric acid and made up to 100 millilitres. For titration 10 millilitres of the stock solution were diluted to 100 millilitres.

All reagents were made up freshly as required with glass-distilled water and kept in brown bottles in a refrigerator.

The dye solution was standardized every day according to the method of Bessey and King[25] with pure ascorbic acid solution, pH 1·5 to 2·0. (According to King,[26] the pH of the end-point should be kept below 3·5 to obtain satisfactory results.)

The Mohr's salt solution was in turn standardized every day against the dye, as described by Lorenz and Arnold.[27]

Technique.

To five millilitres of milk (in duplicate) five millilitres of metaphosphoric acid were added and titrated with the dye solution from a microburette till the appearance of the first pink colour. An excess of dye was then added and the back titration with Mohr's salt solution was carried out drop by drop till the pink colour completely disappeared. As the Mohr's salt solution was prepared so that 10 millilitres corresponded to three to four millilitres of dye, the accuracy of estimating the end-point was greatly increased.

[1] To estimate the loss of vitamin *C* caused by cool storage, several samples were mixed with equal volume of a 3% solution of metaphosphoric acid, placed in a blackened, evacuated desiccator, and left for twenty-four hours in a refrigerator. Under these conditions the loss of vitamin *C* was from 2% to 7%.

[2] As the full description of Kuhn's method was unobtainable, the details of this modification had to be worked out by the writer.

TABLE II.

Variations in Ascorbic Acid Content of Different Portions of the Same Milk. (*Milligrammes* per Centum.)

Type of Milk.	Number of Sample.								
	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.
First milk	0·84	4·02	6·15	1·58	6·52	3·67	5·22	3·95	3·35
Middle milk	0·78	3·68	—	1·55	6·57	3·65	5·78	3·73	—
End milk	0·79	3·66	5·93	1·49	6·49	—	—	3·70	3·42

Preliminary Work.

As many authors point out, there are great variations in the ascorbic acid content of milk occurring one day and from day to day. Differences have also been found in milk from different breasts and in various portions of the same milk. In view of this variability preliminary tests were made to ascertain whether the method of collecting and sampling caused any additional errors.

Nine milk samples were taken in two or three portions, as the first, the middle, and the end milk. The results given in Table II show certain fluctuations; but the statement by Schlemmer *et alii*[28] that the end milk is richer in vitamin *C* than the first or middle milk was not substantiated. There was also no significant difference between portions of milk taken from different breasts, as is indicated in Table III.

TABLE III.

Variation in Ascorbic Acid Content of Milk from Different Breasts (Milligrammes per Centum.)

Breast.	Sample.			
	I.	II.	III.	IV.
Left	4·02	6·57	4·08	3·53
Right	4·19	6·49	4·12	3·65

The day-to-day variations in individual samples were considerable, but they were much smaller when taken as averages of a number of samples (see Table IV). These samples were taken during the colostral period between the fifth and tenth days. Apart from the results on the sixth day the differences were small. Selleg and King[29] obtained similar results.

TABLE IV.

Day-to-day Variations Taken on a Number of Samples (Colostral Period).

Day *Post Partum.*	Mean Value of Ascorbic Acid. (Milligrammes *per Centum.*)	Number of Samples.
5th	3·91	43
6th	3·41	45
7th	4·13	47
8th	4·12	29
9th	4·13	17
10th	3·79	22

Main Investigation.

Over 500 samples of milk from normal and abnormal women were examined. They included (i) colostral and (ii) mature milk.

Colostral Period.

Two hundred and eight samples from 186 normal mothers were examined between the fifth and tenth days *post partum*. From some of the patients several samples were taken on different days, always at the same feeding time.

The ascorbic acid content of milk ranged from 0·86 to 7·40 milligrammes *per centum*, the mean value being 3·87 milligrammes *per centum* and the standard deviation being 1·45. The frequency distribution curve had a normal appearance.

Fifty samples from 36 mothers suffering from toxæmia, albuminuria or post-partum eclampsia and from women whose babies had been born prematurely, were taken between the third and eleventh days after confinement. The mean value, which was 3·94 milligrammes *per centum* for all cases, and 4·00 milligrammes *per centum* for the 23 cases of prematurity, did not differ greatly from the mean for normal cases (contrary to Elmby[11] and Correns,[30] who investigated only a small number of cases). The results did not include the figures of 9·37 milligrammes *per centum* and 6·30 milligrammes *per centum* (average of three estimations) obtained from two patients kept on a diet rich in vitamin *C*.

Mature Milk.

Samples numbering 261 from 186 normal mothers were taken at intervals of one month throughout the whole remaining period of lactation. The ascorbic acid content ranged from 0·39 to 7·74 milligrammes *per centum*. The mean value for the whole period was 3·67 milligrammes *per centum*. A single sample with ascorbic acid content of 9·37 milligrammes *per centum* (see above) was not included. The frequency distribution curve had a normal appearance.

The mean value for individual months of lactation (see Table V) rose slightly from the colostral period, remained constant during the first and second months, then gradually fell, and after reaching its lowest level during the sixth month, slowly rose again and regained its former level during the ninth and tenth months. Although its range of variations (2·99 to 4·01 milligrammes *per centum*) was small in comparison with that of individual samples, the results were statistically examined by the application of the t-test. The mean values for each of the first two months were identical. The differences between these values and those for each successive month up to the sixth proved to be statistically significant. This indicated a fall in ascorbic acid value of milk with the progress of lactation. The seasonal variations could not be mainly responsible for this fall, as in every group of samples taken during the same stage of lactation several were taken during each of the calendar months of spring and summer. The rise after the sixth month could be explained by the fact that the child, being put on "educational diet", consumed less breast milk, and the concentration of ascorbic acid rose with the decrease in volume secreted, which automatically followed the smaller intake by the child.

The mean value for colostral milk was slightly higher than that for the mature milk taken as a whole, but not in comparison with each individual month of lactation. Among overseas workers, Bleyer,[31] Schlemmer *et alii*,[28] Kasahara and Kawashima,[32] Wachholder,[7] de Haas and Meulemans[33] and Stoerr[34] found the ascorbic acid content higher in colostrum than in mature milk, while Neuweiler[35] and Winkler and Heins[36] could find no difference in the two values. Schlemmer *et alii*,[28] Kasahara and Kawashima,[32] and Wachholder and Correns[30] noticed a fall in ascorbic acid value of milk while the lactation proceeded. Bleyer,[31] Neuweiler,[35] Winkler and Heins,[36] on the other hand, consider the level to be constant, provided that there is no vitamin *C* deficiency in the mother's diet.

TABLE V.

The Ascorbic Acid Content of Milk According to the Month of Lactation.

	Month.												
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th	13th
Ascorbic acid (milligrammes *per centum*)	4·00	4·01	3·31	3·12	3·28	2·99	3·37	3·21	4·03	3·91	3·60	4·76	4·59
Number of samples	45	48	37	30	20	18	11	14	9	8	6	5	1
Standard deviation	1·60	1·69	1·33	1·21	1·04	1·11	1·23	1·13	1·39	1·00	1·60	—	—

Seasonal Changes.

To obtain a picture of the seasonal fluctuation the results were regrouped according to the calendar month. Colostral and mature milk, however, was treated separately as representing different time intervals in lactation periods (see Tables VI and VII).

TABLE VI.
The Ascorbic Acid Content of Colostral Milk During the Spring and Summer Months.

Month.	Ascorbic Acid. (Milligrammes *per Centum*.)	Number of Samples.
September ..	4·41	50
October ..	3·51	54
November ..	4·18	65
December ..	4·00	24
January ..	4·10	23

TABLE VII.
The Ascorbic Acid Content of Mature Milk During the Spring and Summer Months.

Month.	Ascorbic Acid. (Milligrammes *per Centum*.)	Number of Samples.
September ..	4·12	47
October ..	3·68	33
November ..	3·28	38
December ..	3·82	32
January ..	3·75	37
February ..	3·56	71

The ascorbic acid content was lowest in October for colostral milk and in November for mature milk, while in both it was highest in September. The colostral period, which represented a large number of samples taken at the same stage of lactation, was chosen for statistical analysis. This revealed a significant difference between the mean values for September and October and for October and November, while the differences between the other months were not statistically significant.

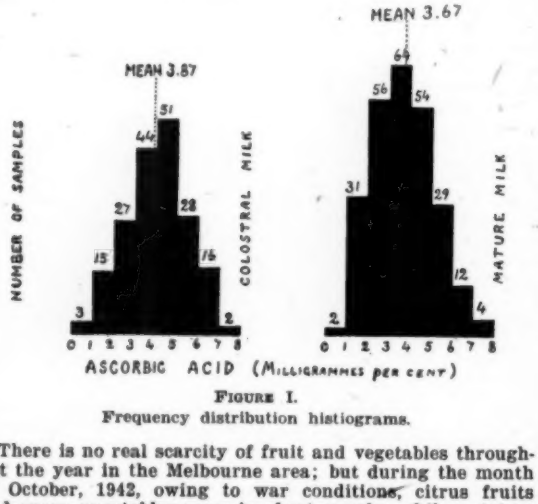

FIGURE I.
Frequency distribution histiograms.

There is no real scarcity of fruit and vegetables throughout the year in the Melbourne area; but during the month of October, 1942, owing to war conditions, citrus fruits and green vegetables were in short supply, while potatoes were not available until the middle of November, when the new crop arrived. This coincided with the significant drop in ascorbic acid value. Although no estimations were made during the winter months in the course of this investigation, it might be expected that under normal conditions the values during this period would have fallen to the level of October, 1942, or below.

Discussion and Conclusion.

During the last decade numerous publications have appeared in the scientific literature concerning the vitamin *C* content of breast milk in different countries. Unfortunately, owing to war conditions, many of these publications have been available only by title or in the form of short abstracts. Furthermore, as many of the authors based their results on a small number of samples, only those publications were selected which could give reliable information on the vitamin *C* level of breast milk in their respective countries.

The results are summarized below.

Colostral Milk.

The figures recorded were as follows: Selleg and King,[76] 5·5 milligrammes *per centum* (53 samples); Gaehtgens and Werner,[135] 1·8 to 7·6 milligrammes *per centum* (set groups of 10 to 20 samples); Kasahara and Kawashima,[32] 7·0 to 8·0 milligrammes *per centum* (122 samples).

Mature Milk.

The figures recorded were as follows: Harris and Ray[37] (London), 5·6 milligrammes *per centum* (135 samples); Kasahara and Kawashima[32] (Japan), 4·5 milligrammes *per centum* ("a number"); Laurin[17] (Sweden), 3·0 to 7·0 milligrammes *per centum* (55 samples); Gedd and Kjelberg[14] (Gottenburg), 3·9 milligrammes *per centum* (421 samples); Ingalls *et alii*[20] (United States of America), 4·5 milligrammes *per centum* ("a number"); El-Gholmy[40] (Cairo), 3·8 milligrammes *per centum* (140 samples); Isono[41] (Japan), 3·5 milligrammes *per centum* (57 samples).

In addition, a few other publications were selected in which the results, although based on a small number of samples or given without the number of samples examined, were nevertheless of some interest. They are as follows: Braëstrup and Lieck[42] (Copenhagen), 2·0 to 6·0 milligrammes *per centum*; Traversaro and Quesada[43] (Argentina), 5·0 milligrammes *per centum* (six samples); de Haas and Meulemans[22] (Batavia), 4·0 to 5·0 milligrammes *per centum* (native women), 6·0 to 7·0 milligrammes *per centum* (European women); Cimmino[44] (Italy), 2·7 milligrammes (five samples); Concepcion and Gargaritano[45] (Philippines), 4·0 to 5·0 milligrammes *per centum*; Sapegno and Mado[46] (Turin), 4·0 to 5·0 milligrammes *per centum*; Chakraborty[47] (Calcutta), 5·0 milligrammes *per centum*; Kroker[48] and Elmby,[11] who give respectively 2·2 to 4·2 milligrammes *per centum* for low income groups and 8·7 milligrammes and over 7·0 milligrammes *per centum* for high income groups.

Seasonal Changes.

All authors agree that the vitamin *C* value of milk undergoes certain seasonal variations, particularly in countries where there is a shortage of fresh fruit and vegetables in winter-time. Sinkko,[49] in Finland, who investigated the ascorbic acid content of the milk of ten women throughout the year, found that it was low in winter-time. High values were obtained in August and September, but there was a fall in October and November; the lowest value of 1·76 milligrammes *per centum* was reached in February, as compared with 4·63 milligrammes *per centum* in September. The variations in the infant mortality rate during the year are correlated by Sinkko with the seasonal variations in the content of breast milk. Winkler and Heins[20] found the values to be 4·6 to 6·6 milligrammes *per centum* in summer and 2·9 to 5·2 milligrammes *per centum* in winter, and Gedd and Kjelberg[14] (Gottenburg) found the values to be 4·2 milligrammes *per centum* in autumn and 3·6 milligrammes *per centum* in spring. Baumann[13] (Switzerland) reported for the period from November to January a range of 2·5 to 3·0 milligrammes *per centum*, and for the period from June to October (summer) 4·0 to 7·5 milligrammes *per centum*. He noticed an unexpected rise after January (February to May, 3·5 to 5·7 milligrammes *per centum*) due to the arrival of imported citrus fruit. Ferdinand[9] (Rostock, Germany) found values of 3·5 milligrammes *per centum* for winter and

2·1 milligrammes *per centum* for early spring, while Correns[20] (Rostock) found values of 3·93 milligrammes *per centum* for summer. The results for summer and winter in Rostock were thus not greatly different, owing to the availability of a good supply of food rich in vitamin *C*. The low values for early spring would coincide with the period in central Europe during which fresh greens or other sources of the vitamin are scarce.

Comment.

Compared with most of the reliable overseas reports, the average ascorbic acid content of breast milk in the Melbourne area, ranging from 3·0 to 4·0 milligrammes *per centum* throughout the whole lactation period, is low even during the summer months.

According to Ingalls,[20] Smith[3] and others, in order to assure a daily supply of 20 to 40 milligrammes, which is essential for adequate nutrition of the infant, the ascorbic acid content of milk must not fall below the level of 4·0 milligrammes *per centum*. In the Melbourne area the average value just reached this level only during the first two months of lactation. During the remaining four months until the infant began, or should have begun, to receive the orange juice, the level was found to decline to 2·99 milligrammes *per centum*.

The frequency distribution histograms (Figure I) reveal that in about 50% of cases during the colostral period and in about 60% in the later stages the level was below 4·0 milligrammes *per centum*. This means that more than half of the breast-fed infants in the lower and medium income groups received a diet deficient in vitamin *C* even during the summer months, while those babies receiving pooled mother's milk or cow's milk supplement had an even lower vitamin intake. The histograms also indicate that a large portion of the adult female population had a subclinical vitamin *C* deficiency.

Therefore it seems to be of great importance to give both the mother and the breast-fed infant orange juice or synthetic vitamin *C* as early as the first few weeks after the mother's confinement.

Summary.

1. Preliminary work was carried out as follows. (*a*) Nine milk samples were examined in two or three portions. The end milk was not richer in ascorbic acid than the middle or first milk. (*b*) Four milk samples were taken from both breasts and found to have the same ascorbic acid content within the limits of experimental error. (*c*) A number of samples was studied during the colostral period on six consecutive days. The differences between the averages of a number of samples were much smaller than the day-to-day variations in individual samples.

2. During the colostral period 208 samples from 186 healthy mothers were examined between the fifth and tenth days *post partum*. The ascorbic acid content of milk ranged from 0·86 to 7·40 milligrammes *per centum*, the mean value being 3·87 milligrammes *per centum* and the standard deviation 1·45.

3. Fifty milk samples from 36 mothers suffering from toxæmia, albuminuria and post-partum eclampsia, and from mothers of premature infants, were examined between the third and eleventh days *post partum*. The mean value was 3·94 milligrammes *per centum* for all the women and 4·00 milligrammes *per centum* for the 23 with premature infants. These figures do not differ greatly from the values for normal women.

4. Samples numbering 261 from 186 healthy mothers were taken at intervals of one month during the whole period of lactation. The ascorbic acid content ranged from 0·39 to 7·74 milligrammes *per centum*. The mean value for the whole period was 3·67 milligrammes *per centum*. The mean values for individual months rose slightly from the colostral period, remained constant during the first and second months and gradually fell, reaching the lowest level of 2·99 milligrammes *per centum* during the sixth month, and then rose again.

5. The differences between the third, fourth, fifth and sixth months as compared with the first two months proved to be statistically significant, indicating a fall in ascorbic acid level with the progress of lactation up to the sixth month.

6. The mean value for colostral milk was slightly higher than that for the mature milk taken as a whole, but not in comparison with the figures for each individual month of lactation.

7. The results, regrouped according to the calendar month, have shown a significant fall in vitamin *C* content for colostral milk in October and for mature milk in November, which coincided with the shortage of potatoes and fresh vegetables.

8. The average ascorbic acid content of milk in the Melbourne area, ranging from 3·0 to 4·0 milligrammes *per centum*, was low in comparison with the figures obtained in other countries.

9. The frequency distribution histograms revealed that from 50% to 60% of breast-fed babies in the lower and middle income groups did not receive an adequate amount of vitamin *C* during their first six months of life, as 4·0 milligrammes *per centum* is considered the minimum ascorbic acid concentration in milk necessary for normal nutrition.

10. The writer considers that it is urgent to administer some vitamin *C* supplement even to breast-fed babies as early as the first few weeks of life.

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CHYLANGIOMA OF THE MESENTERY, WITH REPORT OF A CASE, AND A BRIEF DISCUSSION OF MESENTERIC CYSTS.

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A CHYLANGIOMA of the mesentery has been defined by Ewing[1] as a cavernous lymphangioma containing milky fluid, which arises from congenital or acquired obstruction of the lacteal vessels. Chylangioma constitutes one type of true mesenteric cyst, in that it conforms to the definition of a mesenteric cyst given by Higgins and Lloyd,[2] who held that mesenteric cysts were cysts which occurred in or near the mesentery, which were not malignant, dermoid or parasitic, and which did not arise from any normally placed retroperitoneal organ.

Great interest has been shown in the study of mesenteric cysts since the discovery at autopsy, by Benevieni, of the first recorded mesenteric cyst in 1507. The history of the knowledge accumulated in respect to cysts of mesenteric origin is extensively reported; the reviews of Warfield,[3] Swartley,[4] and Roller[5] may be quoted as adequately summarizing the historical aspect.

Of all the abdominal tumours, Flynn[6] considers those of the mesentery to be the rarest. Mesenteric cysts are uncommon, Warfield estimating that only about 500 cases of mesenteric cyst have been reported in the literature. Among these cases, Slocum[7] states that less than 300 true mesenteric cysts have been reported, and that the number of true chylous cysts of the mesentery of sufficient size to cause symptoms would be no greater than 200. Parsons,[8] after reviewing the literature, states that there are records of only ten cases of mesenteric cyst with gross and microscopic appearances of true proliferating cystic lymphangioma, although the criteria on which this assertion is made are not definitely enumerated in the article. It appears to us that the condition is more common than these figures lead one to believe, but, because lymphangiomata (or chylangiomata) are still rare, we venture to report a case of this type of cyst which we regard as a true chylangioma.

The frequency with which mesenteric cysts are found can be judged from a review of the medical literature which contains statistical surveys. At the University of Minnesota, Collins and Berdez[9] found no case of mesenteric cyst in 15,000 autopsies, and only two cases of chyle cysts were found in the clinical records of 200,000 cases. These figures are comparable with the records of

the Mayo Clinic, where Crisp and Judd[10] found the histories of eight cases of cysts amongst 25 mesenteric tumours in 820,000 admissions. Alesen[11] was able to find no case of mesenteric cyst recorded at the Los Angeles General Hospital between 1912 and 1929; but during the following five-year period one case was recorded in 188,921 admissions (Roller[5]). The average statistics, of which a long series has now accumulated, give the relative frequency of mesenteric cysts as approximating to one in every 100,000 admissions.

Review of available Australian literature covering the last twenty years has shown little reference to mesenteric cysts in Australia. Phillips[12] failed to find any case amongst 19,788 admissions to the Alfred Hospital, Melbourne, during the years from 1930 to 1933, or in 4,000 post-mortem examinations performed at that hospital since 1914. Three mesenteric cysts have been recorded in Australian medical journals during this time. In 1927 Lee[13] reported a mesenteric cyst simulating post-partum dilatation of the stomach; in 1928 Wade and Steigrad[14] recorded a case of lymphatic mesenteric cyst; and in 1935 Ross[15] described an enterogenous cyst in an infant.

Recently, at a Royal Australian Air Force hospital, a case of chylangioma, or cavernous lymphangioma containing chyle, was investigated by us and an exploratory operation was carried out. No other case of mesenteric cyst has been recorded in 13,744 admissions to this hospital. Because of the rarity of this type of mesenteric cyst, the case history will be briefly given and can be used as a basis for discussion of mesenteric cysts generally.

Report of a Case.

An airman of the Royal Australian Air Force, A.J.H., aged twenty years, first reported in June, 1944, that he had noticed an epigastric "lump", which was associated with some vague abdominal discomfort. The examining medical officer could feel a tumour mass in the epigastrium, which appeared to have a diameter of three inches. Further questioning elicited a history of three days' persistent umbilical pain, anorexia and nausea, but no vomiting. A curious feature was the complaint of persistent lumbar backache. Inquiry into the past history revealed no significant features. The patient was admitted to hospital at this time with a normal temperature and pulse rate, but with a "dirty" tongue and "heavy" breath. Abdominal examination revealed tenderness above and to the right of the umbilicus, with some abdominal rigidity. At this time the mass could not be palpated, although two days later a cystic, freely movable mass could be felt in the left lower quadrant of the abdomen. Shortly afterwards the tumour apparently disappeared, and repeated palpation gave negative results. An outstanding feature of the cyst has been its free mobility at all times, and a tendency to disappear, probably due to its being moved to a position under cover of the costal arch. The blood film was normal, and there was a slight decrease in the hæmoglobin value (93%). The Casoni test, the hydatid complement fixation test, the Wassermann test, a plain X-ray examination of the abdomen, and an opaque meal examination of the gastro-intestinal tract were all carried out with negative results.

In December, 1944, he was readmitted to hospital with some joint pains, which were considered to be of mild rheumatic nature. Once again the mass was palpable, but, being in the left loin, was considered probably renal in origin. Electrocardiograph tracings, a blood count, and estimation of the blood sedimentation rate gave normal results. X-ray films of the renal tract revealed an enlarged left renal shadow, which was, however, considered to be within normal limits, and no opaque calculi. Excretion pyelography revealed the dye excretion, the renal calyces, the pelvis and the ureter to be normal. After a month in hospital the patient was returned to duty, the mass being indefinitely palpable below the left costal margin and being considered possibly a kidney.

On June 1, 1945, the patient was readmitted to hospital with pain in the upper part of the abdomen and in the lumbar region, identical with that experienced a year previously. He was also aware of a freely mobile epigastric tumour. Clinical examination gave negative results, except for the presence of a smooth, freely movable, cystic mass about four inches in diameter, with regular outline, and situated in the umbilical region. While freely mobile, the mass could most easily be displaced into the right upper quadrant of the abdomen. The clinical diagnosis was between a mesenteric cyst and a hydatid cyst. The Casoni test was repeated with negative results. Microscopic examination of the urine revealed no siginificant features. A blood examination showed that the hæmoglobin value was 12·6 grammes per 100 mils (90%), the red blood cells numbered 4,670,000 per cubic millimetre, and the white blood cells numbered 8,700 per cubic millimetre, 66% being neutrophile polymorphonuclear cells, 23% being lymphocytes, 7% monocytes and 4% eosinophile polymorphonuclear cells.

Operation, performed on June 7, 1945, revealed a large, fleshy, irregularly rounded, well-differentiated tumour arising in the mesentery in the region of the duodeno-jejunal junction and the upper part of the jejunum, and extending towards its posterior attachment, thereby surrounding the superior mesenteric artery. The tumour was mottled, with pale pink and creamy-yellow areas throughout. The mesentery of the jejunum was widely separated by the tumour, which had extended in such a manner that it had formed a gutter that partly surrounded the jejunum over its proximal eight inches. There was no evidence that the tumour had infiltrated surrounding tissues, no obstruction to the lumen of the bowel was evident, although some thickening of the bowel wall over the first ten or twelve inches occurred, and a small number of the lacteals draining the area were thickened, dilated and filled with a material resembling clotted chyle or fat. The mass was of varying consistence, being formed of fleshy masses separated by cystic spaces, varying in size from the size of a pin's head to that of a golf ball; the whole area had a typical spongy appearance. The tumour, which was more extensive than clinical examination had led us to believe, was not invading surrounding structures, but its removal was considered to be technically impossible because of its relationship to the superior mesenteric artery, and marsupialization would not drain the cyst. Aspiration was carried out as a palliative procedure, no gastro-jejunal anastomosis being deemed necessary, as obstruction of the lumen of the jejunum appeared to be a most unlikely development for a considerable time. A biopsy specimen of the tumour mass was taken to confirm the diagnosis of chylous cyst of the mesentery. The abdomen was closed after operation.

The fluid obtained from the cystic spaces was milky, contained fat globules, and was partly pale pink from blood staining. The pathological report on the fluid was that the specimen of fluid was milky and blood-stained, and on microscopic examination was found to contain red blood cells, a few lymphocytes and granular material which resembled fat. The microscopic appearance of the biopsy specimen (see Figure I) revealed adipose tissue, in which

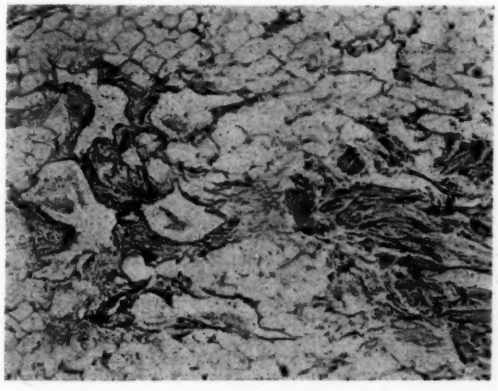

FIGURE I.

Photomicrograph of biopsy specimen of the chylangioma, showing a network of lymph spaces lined by flattened endothelium and surrounded by irregularly arranged, smooth muscle fasciculi. (Hæmotoxylin and eosin stain, × 105.)

some small blood vessels, irregularly shaped spaces and smooth muscle fibres were present. The spaces were lined by flattened endothelium and contained a small quantity of granular eosinophilic material and a few scattered red cells. The smooth muscle was irregularly arranged around these spaces, particularly the larger space visible in the section, and here the fibres tended to be grouped into interweaving fasciculi in the walls of the space. The pathologist's opinion

was that these histological findings, taken in conjunction with the character of the aspirated fluid, indicated the diagnosis of chylangioma.

Convalescence was uneventful, and although a mass, smaller and less mobile than formerly, is still palpable in the lower part of the epigastrium, the patient is symptom-free and has returned to his pre-service occupation.

Discussion.

The classification of mesenteric cysts is confused, and no uniform opinion is noted amongst many schemes propounded. Moynihan in 1897 produced a simple classification, which was succeeded by the classification of Dowd,[16] who stressed the embryonic origin of many cysts. Subsequent classifications have been a modification or amplification of these earlier schemes.

Roller[6] gives the following scheme as a basis for classification of mesenteric cysts:

1. Embryocytomata or embryonic inclusion tumours.
   (a) Urogenital remnants derived from sequestrated portions of Wolffian or Müllerian ducts.
   (b) Cystic dermoids and teratomata resulting from sequestration of remnants of the developing genital glands.
   (c) Dermoid inclusions (very rare).
   (d) Enterocystomata arising from sequestration of either an intestinal diverticulum or the vitelline duct.
2. Chylous cysts, which are considered by Ewing to be chyle angiomata and by Dowd to be chylous secretion into preformed cysts.
3. Bacterial or parasitic cysts, such as occur as cystic formations in tuberculous glands or hydatid cysts.
4. Traumatic cysts, arising from hæmatomata in the mesentery or developing around a foreign body.
5. Angiomata of blood or lymph vessels. The latter would be lymphangiomata and should therefore be classified with the chylous cysts above.
6. Gas cysts, which are included with reservation.

In the classification which Petersen[17] proposes, the primary distinction is made between embryonic cysts and pseudo-cysts, which are grouped thus:

1. Embryonic.
   (a) Cysts arising from embryonic remnants and sequestrated tissue: (i) serous, (ii) chylous, (iii) sanguineous, (iv) dermoid.
   (b) Cysts of intestinal origin: (i) by sequestration from the bowel during development; (ii) arising from Meckel's diverticulum.
   (c) Cysts arising from urogenital organs (germinal epithelium, ovary, Wolffian body or Müllerian duct).
2. Pseudo-cysts.
   (a) Of infective origin.
   (b) Cystic malignant disease.

However, for the purpose of study, and as a basis for discussion, the simple classification of Ewing[1] appears adequate. He concisely divides mesenteric cysts into four groups, as follows: (i) lymphatic or chylous, (ii) enteric, (iii) urogenital, (iv) dermoid and teratoid.

It will be noted that cysts of neighbouring organs, encysted hæmatomata, malignant cysts, infective cysts and hydatid cysts are excluded, and that gas cysts, which are rare, of disputed ætiology, and considered by many to be due to gas formation in dilated lymph or chylous channels, are not classified.

Dermoids and teratoid tumours are extremely rare in the mesentery, although they can occur at any point from the cœliac axis to the pelvis. Their origin is disputed, but two main views are held. Wilms considers that they arise from imperfect closure of embryonic abdominal plates, but others consider that they arise from an ovarian basis. In the mesentery these tumours differ in no way from similar dermoids and teratoids of other situations.

Cysts of urogenital origin are but rarely found amongst mesenteric cysts and form an ill-defined group. In a discussion on retroperitoneal cysts, Handfield-Jones[18] traces the origin of cysts in relation to the embryonic urogenital system. It is quite conceivable that mesenteric cysts could arise by displacement of remnants of the Wolffian system into the developing mesentery. Cysts of urogenital origin are usually large, thin-walled cysts, lined by low columnar epithelium and having a fibrous tissue wall. In the cyst is found a clear, serous fluid, of low specific gravity, and containing pseudo-mucin. Cysts of urogenital origin are difficult to distinguish from cysts of mesocolic origin, to which attention has been drawn by Handfield-Jones, who related their origin to islands of peritoneum which failed to disappear during the process of development and fixation of the colon.

Enteric cysts form a definite group of mesenteric cysts. Dowd[19] focused attention on the origin of mesenteric cysts from remnants of the Wolffian body, but also admitted that some cysts might arise from intestinal diverticula. Evidence is now conclusive that enterogenous cysts arise in this manner. Miller[19] reports a case in which an enteric cyst could be seen developing from the intestinal wall, and there is abundant evidence that intestinal diverticula may arise in the bowel of the embryo. If such a diverticulum, lying between the layers of the mesentery, becomes separated, it readily forms a potential mesenteric cyst. Such a cyst has usually a single cavity and a wall resembling that of the intestine; it even contains layers of smooth muscle, a mucosa with crypts, and a lining of cylindrical, cuboid or stratified epithelium. Evans[20] states that all cysts found in the abdomen, in the thorax or at the umbilicus, having the structure of gut, must have been derived from the primitive intestinal tract and are developmental enterogenous cysts. These cysts can arise from a diverticulum of embryonic entoderm in this manner, but another source is in relation to the vitello-intestinal tract. The situation of the vitello-intestinal tract in relation to the mesentery is subject to considerable variations, and it is not uncommon for remnants of the vitello-intestinal tract to become separated between the layers of the mesentery, and to give rise to typical enteric mesenteric cysts. It appears probable that cysts arising from vitelline duct remnants would be found towards the lower portion of the small intestine, while those arising from the formation of intestinal diverticula would appear at the upper end.

Chylous or lymphatic cysts have caused a large amount of discussion in medical literature. Many authors, including Dowd,[19] Warfield,[3] and Higgins and Lloyd,[2] regard chylous or lymphatic mesenteric cysts as not arising from obstruction of lymphatics, but as accumulations in pre-existing cysts. However, there seems to be considerable reliable evidence that lymphatic or chylous cysts exist as distinct entities and that they are mostly of embryonic origin, although some may be acquired. The former are the true chylangiomata and the latter are due to obstruction to lymphatic vessels. It is, however, not always easy to distinguish between the lymphangiomatous and the lymphangiectatic variety.

Chylous cysts form large, usually multilocular tumours, or sometimes occur as numerous small swellings of the mesentery, the intestine, the omentum or retroperitoneal organs. Chylangioma can be considered as a tumour of lymph vessels, consisting of endothelial cells and connective tissue, both of which take part in the neoplastic process. The condition is considered of embryonic origin, even though signs and symptoms may not be present until adolescence or adult life. The tumour consists of an anastomosing network of lymph spaces supported by thin walls or thick septa, lined by endothelium, with chylous contents or more inspissated fatty material, and often blood-stained. The endothelium lining the cavernous lymph spaces may be flattened, or in places it may be hyperplastic, proliferating and heaped up into layers. In some specimens evidence of the formation of new lymphatic channels can be observed as an outgrowth of endothelium into the connective tissue and its subsequent canalization. It is possible in cysts with tense contents for the endothelial lining to be distributed in a patchy fashion. The walls of the lymph spaces may be thin or thick septa; they are composed of fibrous tissue, in which may be found many small round cells, lymph follicles and dilated lymph

spaces on many occasions. Some elastic tissue and bundles of smooth muscle may be present in the walls, and giant cells are found about fatty detritus. The presence of smooth muscle in the wall, being a feature common to chylangiomata and enteric cysts, may make their recognition difficult.

The contents of chylangiomatous cysts rarely appear to have been analysed or correlated with the contents of mesenteric cysts whose pathology is known. It appears as if all analyses have been made on "typical" mesenteric cysts of unstated ætiology, and no examination of fluid from a chylous lymphangioma (chylangioma) appears to have been made and compared with that of fluid from a lymphangioma containing clear fluid. Higgins and Lloyd[2] state that "typical" fluid from a mesenteric cyst is pale, clear, straw-coloured fluid with a specific gravity of 1015 to 1016, contains large amounts of albumin, and is alkaline in reaction; blood, cell debris and cholesterin are also usually present. Ross and Mead[21] analysed "chyle-like" and amber fluid from mesenteric cysts, but stated that the sample was not chyle, which consists of lymph containing fat globules, but that it consisted of fluid, the milky appearance of which was due to protein loosely bound with lipoid material, particularly lecithin. The composition of the two samples varied little, except in respect to fat and cholesterol; both contained 94·3% water, 5·7% solids and approximately 3·8% total protein. The fat (ether-soluble) content in the chylous fluid was 0·27% as compared with 0·73% in the amber fluid, and the cholesterol content was 0·178% in the chylous fluid as against 0·38% in the amber fluid. Messer[22] analysed the fluid from a chylous cyst in a patient operated upon by Slocum,[7] comparing its composition with that of chyle, chylous effusion and the contents of chylous cysts reported by other authors. Once again considerable disparity exists in the chemical composition, and no constant feature appears in the analyses of the four authors quoted. The fat content varied from 4·5% to 35·8%, and the protein content from 1·97% to 8·51% in the analysis of the mesenteric cystic fluid. In this series it is again uncertain whether the contents of the chylous cysts should be considered as those of chylangiomata. Messer believed the disparity to be due to variation of the degree of effusion or to differential absorption of the stagnant fluid. The particular specimen examined was a creamy fluid, which on standing separated out, a tenacious creamy sludge being produced. One pronounced feature of chylous fluid that should be noted is its resistance to accidental contamination by bacteria; a specimen remains sterile for long periods in the absence of precautions to avoid entry of bacteria. This is rather surprising, in view of the relatively high protein content; it suggests that the fluid has a bacteriostatic effect.

Macroscopically, chylangiomata are well differentiated from surrounding tissues, having a capsule which varies with the size of the cysts, with their situation, and according to whether they are pedunculated or sessile, or whether they project between the layers of the mesentery. The tumour may be a single cyst or a multilocular cyst with cystic spaces of varying size; it may be entirely cystic or partly fleshy.

Chylangiomata are considered benign tumours, whose embryonic origin is stressed by Klemm and Rittner (as quoted by Ewing[11]) and by Murbach, Lewison and Diebert,[23] who pointed out that lymphangiomata occurred most frequently at the primitive centres of origin of lymph tissue in the embryo.

Acquired dilatation of lymphatic vessels of the intestinal tract can give rise to mesenteric cysts, and such dilatations are included in the definition of chylangiomata outlined at the commencement of this paper. That obstruction of lymphatics can give rise to cysts has been challenged on the grounds that the rich anastomosis of lymphatics would not permit this occurrence. However, there are some grounds for the belief that it is possible, and there is no doubt that lymph stasis influences the course of many congenital lymphangiomata and that cystic dilatation does occur in the newly formed lymph vessels of proliferating cystic lymphangiomata. In some cases the dilatation with cyst formation appears to involve chiefly the lymph nodes. Such a condition would be comparable with the condition described by Hill,[24] to which he has given the name of mesenteric chyladenectasis. He noted that, while excessive dilatation of lymph-node sinuses to form multilocular or single cysts was rare, dilatation of mesenteric gland nodes with inspissated chyle was extremely rare. This dilatation, to which the term "chyladenectasis" has been given, has been variously attributed to obstruction of the lymphatics or the lymphatic duct, to infarction of efferent lymph vessels, to obstruction of the thoracic duct and iliac veins simultaneously, to chronic desquamative lymphangitis, and to inflammatory change altering the local lymphatic circulation. Cystic changes are present in the lymph nodes, which have the character of lymph-gland medullary sinuses, with no true endothelial lining, and absence of smooth muscle around the cystic channels. Hill states that chyladenectasis should be considered in cases of multilocular, multiple cysts located in the mesentery, particularly in adults, and not involving the intestinal wall. The characters of these cysts are the presence of chylous or fatty material in dilated lymph-node sinuses without complete endothelial lining, absence of smooth muscle in the cyst walls, and suitable obstruction to chylous drainage.

The differentiation of mesenteric cysts into their various groups is not easy on either anatomical or pathological grounds; but it may be stated that the presence of smooth muscle, epithelial lining of intestinal type and perhaps goblet-cell formation suggests enteric cysts; endothelial lining suggests chylangioma; high columnar epithelium and pseudo-mucin, hair or teeth, suggest origin from primitive sex organ ancestry (Warfield[3]).

Mesenteric cysts occur at any age, but the maximum incidence appears to be in early adult life. There is general agreement on this, although Higgins and Lloyd[2] state that the maximum incidence is between the ages of ten and twenty years, while Warfield[3] found the greatest incidence in the fourth decade, and Collins and Berdez[6] found it in the third decade. The female sex seems to be more commonly affected than the male.

Warfield[3] has stated the distribution of mesenteric cysts reported in the literature in their relation to the intestine; but, unfortunately, no statistics appear to be available as to the location of various pathological types of cysts in relation to the bowel. It was found that cysts of the small intestinal mesentery were slightly more frequent than those of the large bowel; that 1·5% occurred at the duodeno-jejunal junction; that cysts of the ileum were almost three times as common as cysts in the jejunum; and that the three divisions of the colon had approximately an equal number of cysts in relation to them.

The symptoms arising from mesenteric cysts are in no way pathognomonic; it is often the onset of an acute surgical complication that first draws attention to the condition. In uncomplicated cases the patient presents for examination with the complaint of a palpable tumour or of abdominal distension. The tumour varies in size, but characteristically it is smooth, rounded or lobulated, somewhat centrally situated in the abdomen; perhaps its most prominent feature is its extreme mobility, especially in a transverse direction. In favourable circumstances the fluctuant or fluid nature of the cyst contents can be demonstrated. The second important feature which draws the patient's attention to his condition is pain; this is nearly always present, even in the absence of complications which almost invariably are accompanied by severe pain. The pain may be generalized over the abdomen or may be confined to the tumour itself. In the latter case particularly, localized tenderness may be elicited. It is stated that pain is more frequently present in mesenteric cysts than in any other cystic abdominal tumour. The reason for this is not apparent, although the curious lumbar pain in the case quoted was probably due to drag or tension on the mesentery. The site, nature and cause of pain in uncomplicated mesenteric cysts do not appear to have received much attention in the literature.

Other symptoms, such as malaise, anorexia, nausea or occasional vomiting are not prominent features, but are

more common in mesenteric cysts than in those of retroperitoneal regions. Fatigue and loss of weight and strength may occur, but these are only of minor grade; the rapid loss of weight, which Moynihan thought was a feature of chylous cysts, does not appear to be a constant finding.

The classical clinical signs were considered to be the prominence of a fluctuant tumour towards the umbilical region, great mobility in a transverse direction, and the presence of a "zone of resonance around and a belt of resonance across the cyst". The last-mentioned sign must be most inconstant, apparently has no sound logical basis, and is rarely mentioned by modern writers. The features of the palpable tumour have already been mentioned, except that the percussion note over the tumour may be dull, and variable bands of resonance as described above not be present. Tenderness over the cyst is frequently found even in the absence of complications.

The usual history, physical signs and symptoms presented in a case of mesenteric cyst are those of complications which may arise and will be discussed no further.

In a review of cysts of the omentum, of the mesentery, and of retroperitoneal origin, Berger and Rothenberg,[29] reviewing eighteen cases, of which five were cases of mesenteric cyst, found that eight patients had sought treatment for acute surgical conditions, that eight patients complained of a mass only, and that two-thirds of the patients had had pain; four of the five patients with mesenteric cysts had complained of pain and had a palpable tumour also.

The diagnosis of mesenteric cyst is primarily one of correlating the physical signs of a freely mobile cyst in the abdomen with the knowledge that, although they are rare, mesenteric cysts occur and must be considered in a differential diagnosis. Their final diagnosis is made by elimination of other conditions.

In the matter of diagnosis, the intelligent use of X-ray facilities greatly helps in arriving at a correct opinion. Hinkel[30] states that valuable information concerning location of a cyst can be obtained by palpation under an X-ray screen after the administration of barium orally or rectally, or in both ways, noting indentation of viscera, their mobility and other features. A plain X-ray film of the abdomen occasionally outlines the shadow of a mesenteric cyst, particularly if calcification is occurring in its wall. Excretion pyelography establishes the relation of a cyst to the renal tract and is helpful in differential diagnosis. It has been suggested that pneumoperitoneum methods may help in the localization and identification of a mesenteric cyst.

The differential diagnosis of mesenteric cysts is from ovarian cysts, retroperitoneal tumours, pancreatic cysts, hydronephrosis, hydatid cysts, bowel tumours, pregnant uterus *et cetera*.

Complications which may occur in mesenteric cysts are numerous and give rise to serious conditions whose signs and symptoms overshadow those of the original cyst and for which treatment of a surgical nature is urgently required. Briefly, the complications may be stated as follows.

1. Intestinal obstruction, which occurs in 30% to 50% of cases and has a high mortality rate. The mechanism of the obstruction is variable, symptoms arising from volvulus, angulation of the bowel, adhesions, intussusception, or stenosis of the bowel by pressure from the cyst.

2. Peritonitis, which may develop as a complication of intestinal obstruction or of rupture of the cyst.

3. Hæmorrhage into a mesenteric cyst, which may have acute symptoms and prove fatal. It is also common for a minor grade of hæmorrhage to occur into a cyst, particularly of the chylangiomatous type, giving rise to the pink colour of the contents, but to no other symptoms.

4. Rupture of the cyst, either spontaneously or, more frequently, as a result of trauma of minor or gross degree.

5. Torsion, which may occur, but is infrequent.

6. Incarceration of a cyst in the pelvis; this has been mentioned, but must be a rare complication.

The question of malignant changes occurring in mesenteric cysts is an important one, but malignancy appears to be exceedingly rare. It may, therefore, be considered that mesenteric cysts are benign tumours, and the prognosis will depend upon the size, the location and the site of attachment of the cyst, upon the presence of complications, such as intestinal obstruction and peritonitis, with their high mortality, and, finally, upon the type of operative procedure necessary for the treatment of the condition. The malignant changes that have been described are of a sarcomatous or carcinomatous nature, but very few cases indeed have been reported.

The treatment of mesenteric cysts, without complications, has become standardized and can be considered in four sections, as follows.

1. Enucleation of the cyst is without doubt the ideal procedure. It is most frequently employed and is accompanied by the lowest mortality rate of any curative procedure. Dense adhesions render the operation difficult, and at all times care must be taken to avoid injury to the vascular channels to the bowel.

2. In many instances it is found that enucleation is impossible because of adhesions to the bowel wall, the situation of the tumour, or liability of a loop of bowel to gangrene from damage to vessels in the mesentery. In such cases resection of the tumour and a portion of the bowel is the method of treatment. The mortality rate of this procedure is greater than that of the enucleation operation and is higher still in the presence of complications.

3. In certain circumstances it is impossible to perform either of these operations, and so opening, drainage and marsupialization of the cyst to the abdominal wall are the only feasible procedures. Despite many objections to this method, it has a place in the treatment of mesenteric cysts, it is fairly safe and, given sufficient time, it results in the obliteration of the cyst with final cure. The objections on the grounds of persistent sinus formation, non-obliteration of the cavity and the greater incidence of intestinal obstruction, are more theoretical than real.

4. Aspiration alone is not a satisfactory method of treatment, but it may be used as a preliminary stage in radical surgical treatment. It was used in the case quoted in this paper, with the full realization of the limitations of the method, because radical excision or marsupialization was impossible, and in the absence of obstruction, anastomosis of the bowel was not indicated.

It will be noted that all procedures for relief of the condition are based upon recognition of the benign nature of the usual mesenteric cyst and must be modified in the case of the uncommon malignant cyst.

Cysts complicated by intestinal obstruction, peritonitis, rupture, or the other rarer conditions, primarily demand the standard treatment for these disorders, the decision in respect to the cyst and the line of treatment to be adopted being made at the time of operation. The mortality rate is higher by virtue of the complications, but apart from this fact the treatment is along routine lines and needs no further comment.

Summary.

1. A case of chylangioma of the mesentery at the duodeno-jejunal junction is reported. The cyst had the features of a true tumour of the lacteals, and is sufficiently uncommon to be of interest, especially in view of the infrequency in the literature of reports of mesenteric cysts of any type.

2. The confused classification of mesenteric cysts is discussed, and an attempt has been made briefly to describe common types of mesenteric cysts.

3. The clinical signs, symptoms, diagnosis and treatment of mesenteric cysts are briefly stated.

4. Mesenteric cysts must be more common than appears from perusal of the literature, which, from lack of material, presents an inadequate basis for investigation of the subject. A plea is therefore made for further case reports, so that data can be collected for elucidation of the problem of the nature, frequency and origin of cysts in relation to the peritoneum and mesentery.

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THE TREATMENT OF HÆMOPHILUS INFLUENZÆ MENINGITIS WITH SULPHONAMIDES IN CONJUNCTION WITH HÆMOPHILUS INFLUENZÆ, TYPE B, RABBIT ANTISERUM.

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OF the several forms of infectious meningitis in children, that caused by *Hæmophilus influenzæ* is common and deadly. In spite of the various papers which have appeared on this disease, its importance does not seem to be widely perceived, and so it may be desirable to mention some points about it before discussing its treatment with sulphonamides in conjunction with *Hæmophilus influenzæ* rabbit antiserum, which is the subject of this report.

Frequency.

The frequency of *Hæmophilus influenzæ* meningitis among other kinds of infectious meningitis in children at home and abroad may be gathered from Table I. It is indistinguishable clinically from other kinds of infectious meningitis. The diagnosis is reached as a result of bacteriological examination of the cerebro-spinal fluid, and there is reason to think that *Hæmophilus influenzæ* when present is not always identified. The disease may therefore be commoner than is believed. Suggestions will be made under the heading "Bacteriological Diagnosis" below, which should help to obviate this error.

Age Incidence.

Hæmophilus influenzæ meningitis is about three times more frequent in children aged under two years than in older children (Fothergill and Wright, 1933; Wilkes-Weiss and Huntington, 1936). Of the 388 children who are included in the present report, 68% were aged under two years; the number within each year of age is shown in the bottom line of Table II. The disease is rare in adults.

Mortality.

Alexander's (1943) estimate that, before the introduction of specific treatment, 92% to 100% of affected children died is supported by Neal *et alii* (1934), who reported that four children (3·5%) had recovered out of 111 (all children died out of 62 who were aged less than two years). Alexander's estimate is also supported by Wilkes-Weiss and Huntington (1936), who found among 500 reported cases only nine recoveries out of 373 children aged under two years (2·4% recovered), and 26 recoveries out of 127 aged over two years (20·5% recovered), and by Silverthorne (1943), who reported one recovery among 70 cases. Knouf *et alii* (1942), Neter (1942), and Johnson and Fousek (1943) have reported that all the children in their smaller series (19, 37 and 22 children respectively) died under non-specific treatment. Aléman (1940) found that 15 children (3·2%) recovered out of 478, aged under two years, whose cases were on record on were in her own experience. In the present series two children (1·2%) recovered out of 157 of all ages who received no specific treatment (Table II); they were aged respectively between two and three years and over four years. Children aged twenty-five months or over numbered 50; of these, therefore, 4·0% recovered.

In the case of children aged less than two years, death is so common under expectant treatment that recovery of even a few such children after other treatment may reasonably be attributed to it.

Specific Treatment.

Pittman's (1930, 1931) differentiation of *Hæmophilus influenzæ* into six serological types, *a* to *f*, and her discovery that one type, type *b*, is responsible for practically all cases of meningitis, led to the preparation of type-specific *Hæmophilus influenzæ* horse antiserum. Unfortunately, the clinical results of its use either alone or together with complement (Ward and Wright, 1932; Wright and Ward, 1932; Ward and Fothergill, 1932; Fothergill and Blackfan, 1935) or sulphonamides were disappointing (Pittman, 1933, 1/18;[1] Wilkes-Weiss and Huntington, 1936, 1/19; Fothergill, 1937, 31/201; Lindsay *et alii*, 1940, 9/37; Knouf *et alii*, 1942, 1/32; Silverthorne, 1943, 8/36).

Meanwhile, sulphonamides were found to inhibit the growth of *Hæmophilus influenzæ* *in vitro* and *in vivo* (Pittman, 1939, 1942; Alexander, 1943). Clinical reports

[1] In fractions following authors' names, the denominator indicates the number of children treated, the numerator the number that recovered.

TABLE I.

Relative Frequencies of the Various Kinds of Infectious Meningitis in Children.

Source.	Number of Cases in which Organism was Isolated.							
	Tubercle Bacillus.	Meningococcus.	*Hæmophilus influenzæ.*	Pneumococcus.	Streptococcus.	Staphylococcus.	Others.	Total.
Children's Hospital, Boston, 1920–1931; Fothergill and Sweet (1932)	290	160	78	69	69	—	39	705
Children's Hospital, Boston, 1933–1936; Fothergill (1937)	31	27	51	38	36	—	38	221
Children's Hospital, Washington, D.C. Lindsay, Rice and Selinger (1940)	205	180	100	73	50	5	29	642
Bureau of Laboratories, Department of Health, New York. Neal, Jackson and Applebaum (1934)	961	1,216	111	209	203	27	—	2,727
Hospital for Sick Children, Toronto. Silverthorne (1943)	368	236	153	148	170	25	—	1,100
Royal Alexandra Hospital for Children, Sydney,[1] 1934–1944	120	291	118	86	38	18	78	744

[1] By courtesy of the Medical Superintendent.

TABLE II.

The Numbers of Children According to Age who Received no Specific Treatment (up to 1937), or who were Treated with Sulphonamides (from 1937 Onwards), or with Sulphonamides in Conjunction with Hæmophilus Influenzæ, Type B, Rabbit Antiserum (from 1943 Onwards), and the Numbers who Recovered.

Form of Treatment.	Age in Months.										All Ages.		
	0 to 12		13 to 24		25 to 36		37 to 48		49 and Over.		Total Children Treated.	Number of Recoveries.	Percentage.
	Patients.	Recoveries.	Patients.	Recoveries.	Patients.	Recoveries.	Patients.	Recoveries.	Patients.	Recoveries.			
No specific treatment:													
Royal Alexandra Hospital for Children, Sydney[1]	26	0	30	0	11	0	8	0	9	1	84	1	
Children's Hospital, Melbourne[1]	30	0	22	0	11	1	4	0	6	0	73	1	
Total	56	0	52	0	22	1	12	0	15	1	157	2	1·2
Sulphonamides:													
Royal Alexandra Hospital for Children, Sydney	27	3	22	5	14	6	8	4	15	8	86	26	
Children's Hospital, Melbourne	24	1	24	4	7	0	6	1	3	1	64	7	
Total	51	4	46	9	21	6	14	5	18	9	150	33	22·0
Sulphonamides and serum:													
Royal Alexandra Hospital for Children, Sydney	4	0	1	0	2	1	1	1	2	2	10	4	
Children's Hospital, Melbourne	14	3	16	10	10	8	4	4	0	0	44	25	
Other Australian Hospitals	19	10	4	3	1	1	1	1	2	2	27	17	
Total	37	13	21	13	13	10	6	6	4	4	81	46	56·8
Total	144		119		56		32		37		388		

[1] From figures supplied by courtesy of the medical superintendents.

of its use covered 59 children, of whom 23 (38·9%) recovered (Hamilton and Neff, 1939, 1/1; Sirlin and London, 1940, 1/1; Jacoby, 1941, 1/1; Neter, 1942, 1/3; Knouf *et alii*, 1942, 1/13; Sako *et alii*, 1942, 5/7; Davies, 1943, 4/20; Moir, 1943, 2/4; Hall and Spink, 1943, 2/2; Silverthorne, 1943, 3/5; Cooperstock, 1944, 2/2). Unfortunately, with the exception of Davies's, these reports referred only to single children or to small series of children whose ages were not always given. In a disease in which death under expectant treatment is usual, reports of a new treatment, when based upon small numbers, tend to comprise an unduly high proportion of recoveries; success is more apt to be recorded than failure. Nevertheless, children evidently were saved by sulphonamides, though a recovery rate so high as 38·9% should hardly be expected in statistically adequate series comprising a due proportion of younger in relation to older children. Alexander *et alii* (1942) found that only 12% recovered of 50 children recorded in the literature and from their own experience. Aléman (1940) has reviewed the earlier reports and found 10 patients (12·2%) recovered out of 82 treated with sulphanilamide and five recovered out of 18 treated with sulphapyridine.

Finally, *Hæmophilus influenzæ* rabbit antiserum was introduced by Alexander (1939). In contrast to *Hæmophilus influenzæ* horse antiserum, its efficacy in conjunction with sulphonamides was reported consistently (Neter, 1942, 4/7; Scott and Bruce, 1942, 1/1; Scully and Menten, 1942, 5/9; Grunthal and Winters, 1943, 2/2; Johnson and Fousek, 1943, 17/20; Alexander, 1943, 57/75; Hall and Spink, 1943, 2/2; Silverthorne, 1943, 3/10; Birdsong *et alii*, 1944, 7/8; Boisvert *et alii*, 1944, 22/26; Nicholson, 1944, 2/4; Turner, 1945, 10/20). One hundred and thirty-two children (71·7%) recovered out of the 184 comprised in these reports, most of which are open to the criticisms raised against similar meagre reports of the use of sulphonamides. They include, however, four less inadequate series of 20 or more children, of whom 106 (75·2%) recovered out of 141. Evidently further reduction in the mortality rate followed the addition of *Hæmophilus influenzæ* rabbit antiserum to sulphonamides.

Penicillin is not efficacious in *Hæmophilus influenzæ* infections.

The Present Series.

The data relating to children in the present series who received no specific treatment or who were treated with sulphonamides were provided from the records of the Children's Hospital, Melbourne, and of the Royal Alexandra Hospital for Children, Sydney, by courtesy of the respective medical superintendents. For generous cooperation in supplying all information required for this report we are grateful to them, and also to some fourteen physicians, in the course of whose hospital or private practice in various places children were treated by sulphonamides in conjunction with *Hæmophilus influenzæ*, type *b*, rabbit antiserum.

The serum was prepared in rabbits at the Commonwealth Serum Laboratories against strains of *Hæmophilus influenzæ*, type *b*, isolated from cerebro-spinal fluid. It was treated to reduce its toxicity to human beings (Goodner *et alii*, 1937), but it was not concentrated; it was estimated to contain not less than one milligramme of antibody nitrogen per ml (Alexander and Heidelberger, 1940) on the basis of its precipitin titre and by mouse protection tests (Seidman *et alii*, 1942). It was issued to any physician who required it, who used it at his discretion; it was thus submitted to a field trial in unselected patients under ordinary conditions.

In Table II are shown respectively the numbers of children according to age who received no specific treatment (up to 1937) or who were treated with sulphonamides (from 1937 onwards) or with sulphonamides in conjunction with *Hæmophilus influenzæ*, type *b*, rabbit antiserum (from 1943 onwards), and the numbers who recovered. The independent results from the two widely separated hospitals are shown separately. All children are shown in the table, including some who were moribund or were already suffering from hydrocephalus when treatment was begun; no one is omitted; the table therefore includes the four children previously reported from the Children's Hospital, Melbourne, by Nicholson (1944), and the 29 reported by Turner (1945).

Of 157 children who received no specific treatment, two (1·2%) recovered. On the other hand, of 150 who were treated with sulphonamides, 33 (22%) recovered, and of 81 who were treated with sulphonamides in conjunction with *Hæmophilus influenzæ*, type *b*, rabbit antiserum, 46 (56·8%) recovered. The improvement under treatment with sulphonamides, and the further improvement under treatment with sulphonamides in conjunction with rabbit serum is obviously highly significant (P = 0·0000002).

Discussion.

Influence of Age on Prognosis.

The influence of age on prognosis in children who received no specific treatment has been referred to under the heading "Mortality" above. Under treatment with sulphonamides and under treatment with sulphonamides in conjunction with rabbit serum the recovery rate improved with each year of age (Table II). The influence of age on prognosis is more clearly shown in Table III, in which the results of treatment with sulphonamides or with sulphonamides in conjunction with rabbit serum are arranged within the age groups from birth to twenty-four months and twenty-five months or over. Under each plan of treatment the recovery rate is higher in the higher age group.

As is shown in the last column of Table III, there is a highly significant difference in the survival rate among children treated with sulphonamides and serum as compared with those treated with sulphonamides alone. The significance in the under twenty-four months age group is greater than in the higher age group, but this is because the former group is the larger.

If one assumes that the data presented represent a fair valuation of the efficacy of the two methods of treatment, the addition of serum is capable of saving the lives of about 30 out of 100 children whose ages vary from birth to twenty-four months, of almost 50 out of 100 children aged twenty-five months or over, and of 35 out of 100 children in both groups combined, all of whom would die if treated with sulphonamides alone.

A difference is noticeable in the results obtained in the two hospitals in children aged twenty-five months or more who were treated with sulphonamides. This is the only difference of any significance (P lies between 0·02 and 0·05) noticeable in the results obtained in the two hospitals; in all other groups their results were similar. It seems to provide a further example of the better prognosis in older as compared with younger children, since it is probably to be explained, at least in part, by the preponderance in the Sydney group as compared with the Melbourne group of children who were aged forty-nine months or over (Table II)—15 out of 37 in Sydney as compared with three out of 16 in Melbourne.

Principles and Practice of Treatment.

The disease, though usually fatal, varies much in intensity. It may progress slowly for weeks or appear so suddenly (Hertzog *et alii*, 1944) as to give little opportunity for treatment.

Experimental and clinical evidence suggests that, though sulphonamides inhibit *Hæmophilus influenzæ*, recovery

TABLE III.

The Numbers of Children who were Treated with Sulphonamides or with Sulphonamides in Conjunction with Hæmophilus influenzæ, Type B, Rabbit Antiserum, and the Numbers who Recovered within the Age-Groups 0 to 24 Months and 25 Months and Over.

Age in Months.	Sulphonamides.							Sulphonamides and Serum.									Probability Factor (P).[1]
	Royal Alexandra Hospital for Children, Sydney.		Children's Hospital, Melbourne.		Total Number of Patients Treated.	Total Number of Recoveries.		Royal Alexandra Hospital for Children, Sydney.		Children's Hospital, Melbourne.		Other Australian Hospitals.		Total Number of Patients Treated.	Total Number of Recoveries.		
	Patients.	Recoveries.	Patients.	Recoveries.		Number.	Percentage.	Patients.	Recoveries.	Patients.	Recoveries.	Patients.	Recoveries.		Number.	Percentage.	
0 to 24 ..	49	8	48	5	97	13	13·4	5	0	30	13	23	13	58	26	44·8	0·00002
25 and over	37	18	16	2	53	20	37·7	5	4	14	12	4	4	23	20	86·9	0·00007
Total ..	86	26	64	7	150	33	22·0	10	4	44	25	27	17	81	46	56·8	0·0000002

[1] When P = 0·01, the odds are one in 100 that the difference in results (whatever that may be) is due to chance. Values of 0·05 or less are here regarded as statistically significant, and values of 0·005 or less as highly significant.

depends essentially upon immunity, that is, domination of antibody over antigen. The antibody is either produced naturally or injected therapeutically. The principle of treatment, therefore, as recognized by Alexander and her associates (1942, 1943) is to inhibit invasion by means of one of the sulphonamides until a bacteriological diagnosis has been reached and specific antibody can be injected sufficient to reinforce whatever natural antibody may be produced.

For a discussion of the principles and practice of modern treatment, the reader is referred to the papers just mentioned, on which many of the following remarks are based. Alexander attributes any improvement in her results which may have occurred since the experimental year 1938-1939 chiefly to careful control of the concentration of sulphonamide and antibody in the patient's blood.

The dose of serum, which is expressed in milligrammes of antibody nitrogen, should correspond to the severity of the infection as judged by the glucose content of the cerebro-spinal fluid or by the concentration of specific polysaccharide (antigen) in the body fluids. Alexander gives a good rough-and-ready method of estimating the glucose content of the cerebro-spinal fluid for this purpose: to one mil of Benedict's solution in each of five test tubes are added the quantities of cerebro-spinal fluid shown in Table IV, the concentration of glucose in the cerebro-spinal fluid being estimated according to the volume of fluid required to reduce the Benedict's solution.

TABLE IV.

A Qualitative Test for Estimating the Amount of Sugar in Cerebro-Spinal Fluid in Milligrammes per Centum. (*Alexander et alii*, 1942: *Alexander*, 1943.)

Tube Number.	Mil of Cerebro-Spinal Fluid.	Reduction of Benedict's Solution (1·0 Mil).					
I	0·05	+[1]	0[1]	0	0	0	0
II	0·1	+	+	0	0	0	0
III	0·15	+	+	+	0	0	0
IV	0·2	+	+	+	+	0	0
V	0·25	+	+	+	+	+	0
Milligrammes *per centum* of sugar		Over 50	40 to 50	30 to 40	20 to 30	10 to 20	10

[1] "+" = Reduction of Benedict's solution; "0" = no reduction of Benedict's solution.

The concentration of specific polysaccharide (precipitinogen) is estimated by observing the time taken for the precipitate to appear when cerebro-spinal fluid, clarified if necessary by centrifugation, is carefully layered over *Hæmophilus influenzæ* antiserum in a test tube of small (three to five millimetre) calibre; its appearance within ten minutes is held by Alexander to indicate severe infection requiring a correspondingly large dose of serum. Alexander's schedule of doses in relation to the glucose content of the cerebro-spinal fluid is as follows:

Spinal-Fluid Sugar, Mg. per 100 c.c.	*Antibody Nitrogen Indicated, Mg.*
< 15	100
15 to 25	75
25 to 40	50
Over 40	25

In the present series most of the children who recovered after intravenous injection of the serum were given from 30 to 180 mils, the average total amount being about 130 mils, which was rarely given in more than two doses. As has already been mentioned, the serum was not concentrated.

On the other hand, the seven infants aged under two years (average, eleven months) who recovered after intramuscular administration of serum (see Table V) received an average total dose of 90 mils (minimum 30 mils, maximum 120 mils), the greatest amount given at any one time being 60 mils (to three patients).

Twelve hours after injection of the serum, which Alexander recommends should be given intravenously along with continuous infusion of glucose solution, tests are applied to ascertain whether its dose was sufficient to ensure circulation of free antibody. During the first week enough free antibody should be present in the patient's serum, when this is diluted 1 in 10 and added to a suspension of *Hæmophilus influenzæ*, to cause capsular swelling. Organisms present in the cerebro-spinal fluid (in which they may be preserved by the addition of formalin, 0·4%), if numerous enough, may be used for this purpose, or a young four to six hours' culture in Levinthal broth can be used if organisms are not available in sufficient numbers in the cerebro-spinal fluid. We have had little experience of this use of the Quellung reaction, but we believe that in hands less practised than those of Alexander and her colleagues it may be misleading. At the Children's Hospital, Melbourne, before the decision is made to give a further dose of serum, lumbar puncture is generally performed and attention is paid among other signs to the glucose content of the cerebro-spinal fluid; a low glucose content suggests that a further dose of serum is necessary.

Boisvert *et alii* (1944) report that they have injected the serum intramuscularly into five children, four of whom, all aged less than twenty-one months, recovered. In the present series the numbers within the age groups from birth to twenty-four months and twenty-five months or over who were given the serum intravenously, intramuscularly or by both routes are shown in Table V, in which the better recovery rate after intramuscular injection in the age group from birth to twenty-four months is significant ($P = 0·033$). It may be thought that the serum was given intramuscularly to such children as were not acutely ill, and that it was given intravenously to those who were acutely or desperately ill. The clinical histories do not suggest that this was so; the disease was described by physicians as severe in six of the seven children who recovered in the age group from birth to twenty-four months. No fixed rule can be formed on the evidence available. To some children it would seem imperative to give the serum intravenously. To others a delay of four to six hours before maximum concentration of circulating antibody is reached would not seem to be of crucial importance. It may well be that an initial dose of serum intravenously followed up by a further amount intramuscularly will be found the most generally efficacious. Intrathecal injection of the serum is not recommended.

Alexander attributes the disappointing results of treatment in infants, particularly those aged under seven months, to late diagnosis. Meningitis is harder to recognize early in infants than in older children whose calvaria are rigid, and the physician may be reluctant to submit to lumbar puncture a baby who shows none of the textbook signs of meningitis. It is probably noteworthy, too, that the blood of young children after the age of two months lacks bactericidal power against *Hæmophilus influenzæ*, which is not usually demonstrable until after the third year (Fothergill and Wright, 1933). To judge from the well-known difficulty of immunizing young children, such natural antibody is not readily provoked in young children in response to infection. The delay in beginning specific treatment which results from late diagnosis may well be more dangerous therefore to young than to older children.

Bacteriological Diagnosis.

The following suggestions are put forward, especially for those in remote country districts, in the hope that they will facilitate the rapid identification of *Hæmophilus influenzæ*, type *b*.

As soon as meningitis is suspected, one of the sulphonamides should be given, lumbar puncture should be performed, and a Gram-stained smear should be prepared from a portion of the centrifuged deposit of cerebro-spinal fluid. If small Gram-negative rods or cocco-bacilli are found, a presumptive diagnosis of influenzal meningitis may be made and serum given. The identity of the organism may be tested by the Quellung reaction, although

TABLE V.

The Number of Children Treated with Sulphonamides in Conjunction with Hæmophilus influenzæ, Type B, Rabbit Antiserum, into Whom the Serum was Injected Intravenously or Intramuscularly or by Both Routes, and the Numbers who Recovered within the Age-groups 0 to 24 Months and 25 Months and Over.

Age in Months.	Intravenous Administration.		Intramuscular Administration.		Both Intravenous and Intramuscular Administration.		Probability Factor: Intramuscular *versus* Intravenous Administration.
	Patients.	Recoveries.	Patients.	Recoveries.	Patients.	Recoveries.	
0 to 24	45	17	9	7	4	2	0·033
25 and over	17	15	4	3	2	2	0·5
Total ..	62	32	13	10	6	4	0·063

this is not advised in the case of those without experience of the technique.

The diagnosis may be confirmed within a few minutes in severe cases by the carrying out of a precipitin test on the clear supernatant fluid (Fothergill, 1937), as described above. Failure to elicit this reaction does not, however, exclude influenzal meningitis, and an attempt should always be made to grow the organisms from the remainder of the centrifuged deposit.

The only easily prepared medium on which *Hæmophilus influenzæ* grows freely is chocolate (cooked blood) agar (it grows very poorly on blood agar and ascitic agar). Chocolate agar, when prepared as follows, gives an early and abundant growth. Two mils of red blood cells from defibrinated horse blood (after the supernatant serum has been decanted) are added to 15 mils of melted nutrient agar at 56° C. and mixed, and the whole is heated for ten minutes at 78° C. The medium is then poured into plates and allowed to cool before it is inoculated from the remaining portion of the centrifuged deposit. If growth occurs, there is sufficient within twenty-four hours on which to carry out confirmatory tests, or the culture may be forwarded to a bacteriological laboratory for identification.

Three points are worth remembering: firstly, *Hæmophilus influenzæ* is a common cause of meningitis in young children, and influenzal meningitis is almost invariably associated with one serological type, type *b*; secondly, *Hæmophilus influenzæ* will not grow freely on any of the more commonly used media except chocolate agar; and thirdly, although it grows luxuriantly on this medium, it will die within perhaps four days or so if not sub-cultured.

In such a grievous disease as *Hæmophilus influenzæ* meningitis, even the moderate success here recorded is heartening. It should encourage the physician to continue treatment with one of the sulphonamides, which was probably begun on first suspicion of infection, and urgently to seek a bacteriological diagnosis in order that he may immediately reinforce whatever natural antibody may be present by injecting specific artificial antibody contained in rabbit serum.

Summary.

1. *Hæmophilus influenzæ* meningitis is usually third to tuberculous and meningococcal meningitis among other kinds of infectious meningitis in children. It is about three times more frequent in children aged under two years than in older children. It is rare in adults. Nearly all children aged under two years who receive no specific treatment die; a few older children recover (in the present series 4% aged over two years). Practically all cases are due to one serological type of *Hæmophilus influenzæ*, type *b*.

2. Of 157 children of all ages who received no specific treatment, two (1·2%) recovered; of 150 who were treated with sulphonamides, 32 (22·0%) recovered); of 81 who were treated with sulphonamides in conjunction with *Hæmophilus influenzæ*, type *b*, rabbit antiserum, 46 (56·8%) recovered.

3. Recovery under treatment was more frequent with each year of age. The worse prognosis in very young children was probably due to delay in the commencement of specific treatment resulting from late diagnosis, in consequence of the difficulty of recognizing meningitis early in very young children without resorting to lumbar puncture. It was possibly due also to the absence of natural antibody from the blood of young children and to their relatively poor immune response to infection.

4. One of the sulphonamides should be given. A bacteriological diagnosis should be urgently sought, and specific antibody contained in rabbit serum should be injected immediately the diagnosis is reached.

Acknowledgements.

Our indebtedness to members of the staff of different hospitals, both honorary physicians and other workers, has already been acknowledged in the text. We also wish to acknowledge the advice and help of Mr. J. J. Graydon, M.Sc., of these laboratories, particularly in assessing the statistical values of the results obtained. Finally, we desire to record our appreciation of the skill and enthusiasm of members of the staff of these laboratories, particularly of Mrs. M. Green and Miss O. Coe, without whose wholehearted cooperation sufficient serum for these clinical trials could not have been produced.

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A REPORT ON SIXTEEN CASES OF SUPRAGLOTTIC ŒDEMA.

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In 1941, Sinclair[1] described ten cases of acute laryngitis caused by *Hæmophilus influenzæ* and associated with bacteriæmia. In 1943, du Bois and C. Anderson Aldrich[2] reported four similar cases. It is the purpose of this paper to describe sixteen cases occurring at the Queen's Memorial Infectious Diseases Hospital, Fairfield, over a period of eighteen months during 1944 and 1945, the general clinical picture of which was similar to that described by the above writers. There were, however, certain variations to which reference will be made.

Fifteen of the patients were children, the youngest being aged two years and the oldest ten years. The remaining patient was an adult male, aged thirty-three years. There were two deaths, one of which occurred in the ambulance while the patient was on the way to hospital. All patients except one were examined within twelve hours of the onset of illness, and all were seriously ill with signs of severe respiratory obstruction. In nine instances tracheotomy was necessary, and in three a complicating purulent meningitis developed after a short interval.

The main clinical features were as follows.

1. There was a sudden onset of difficult, obstructed breathing in a previously well child. Grave symptoms developed within a few hours of the onset. There appeared to be no doubt that, in the cases at present under review, the obstruction was supraglottic and not subglottic, and was caused solely by enormous swelling of the epiglottis and aryepiglottic folds. This swelling was so great as to reduce the glottic opening to a narrow slit. There was no true laryngeal stridor, the voice was not hoarse, and the cough when present was not croupy. Both inspiration and expiration were difficult; but the expiratory obstruction appeared to be the greater. Expiration was always accompanied by a sound like a deep-seated snore. It was present in all cases and was considered to be a sign of great value in the diagnosis.

2. Prostration was always a prominent feature. Sinclair described the condition as "shock" for want of a better word. He makes the following statement: "This appearance of shock seemed out of all proportion to the relatively short duration of the obstructive symptoms." A recent article suggests an explanation for this prostration. Saphir reports five fatal cases of laryngeal œdema in which acute myocarditis was found at autopsy.[3]

3. The swollen, hard epiglottis and aryepiglottic folds could be easily palpated by passing a finger down the throat. Direct laryngoscopy was never necessary to confirm the diagnosis.

The history of a typical case is as follows:

A boy, L.H., aged four years, was admitted to hospital with a history of difficult breathing of three hours' duration. Other members of the household had suffered colds, but this child had been well prior to the attack. No previous attacks of asthma or other allergic signs had been noted.

On his admission to hospital, the child was pale and listless. A pronounced expiratory snore was noted. The voice was not hoarse, but it had a slightly "strangled" character. There was a slight cough, but it was not croupy. The throat was reddened, but the fauces were not swollen. A view of the swollen and congested epiglottis could be obtained by the use of the tongue depressor, and the diagnosis was confirmed by digital examination. Two hours after his admission to hospital—that is, five hours after the onset—prostration became more marked, pallor had increased and slight cyanosis of the lips was evident. Tracheotomy was performed under local anæsthesia. During the operation respiration ceased, necessitating hurried opening of the trachea and artificial respiration. A small amount of mucopurulent secretion was then expelled. The epithelial surface

of the trachea appeared normal. Breathing was greatly relieved and the colour improved. Prostration persisted for twenty-four hours, and the child was disinclined to swallow fluids. His temperature on admission to hospital was 103° F. This subsided gradually, but did not return to normal for seven days. By the fourth day the œdema of the epiglottis had subsided sufficiently to permit removal of the tracheotomy tube. Recovery was then uneventful.

The only adult patient, a man, aged thirty-three years, was able to give a clear account of the symptoms of onset.

At the evening meal he noticed some pain on swallowing. His own description was that there was a painful lump in the middle of the throat. This appeared to become larger and more painful during the evening until it affected his breathing. He spent a restless night and at times became very distressed. He was admitted to hospital early the next morning. The clinical picture was typical, but pain was a more prominent feature and prostration was not so pronounced as in the case previously described. The cervical glands were tender, and some œdema of the tissues of the neck was present. He survived the dangerous period without operative interference and made an uneventful recovery.

Complications.

Three children developed purulent meningitis after the initial symptoms of obstruction had been relieved. In the first case meningitis was not diagnosed until fourteen days after the onset of illness, although indefinite meningeal symptoms appeared some days earlier. The second case of meningitis was diagnosed ten days and the third seven days after the onset of the obstructive symptoms.

Bacteriology.

Jackson[4] has described a condition called angioneurotic œdema of the glottis, in which the epiglottis is similarly swollen and œdematous. However, the absence of any previous allergic signs and the lack of response to adrenaline would appear to make this an unlikely explanation. The writers previously mentioned[2,3] have shown that the causative organism was *Hæmophilus influenzæ* in the cases reported by them. The positive cultural findings in the blood would appear to be an adequate explanation of the prostration which occurred.

It must be admitted that the bacteriological findings in the present series have not been so happy or conclusive. At the time when most of these cases were investigated the literature on the subject was not known, and no special effort was made to cultivate *Hæmophilus influenzæ* either from the throat or from the blood. Even after the finding of this organism in the cerebro-spinal fluid in two cases, it was thought that it was probably a secondary invader becoming active after the primary lesion.

Cultural examination of throat swabbings as a rule produced a growth of normal inhabitants of the respiratory tract, namely, green streptococci, Gram-negative diplococci and occasionally staphylococci. Swabs of throats were investigated by two other laboratories independently, with similar inconclusive results. An attempt to isolate a virus was also unsuccessful.

However, in view of the conclusive findings of the writers previously mentioned, and of the similarity of the clinical picture, it is reasonable to suggest that *Hæmophilus influenzæ* was associated with the condition. This opinion is strengthened by the fact that two of the three cases of meningitis which developed were due to *Hæmophilus influenzæ* Type B. In the third case a profuse growth of pneumobacillus of Friedländer was obtained from the throat, the trachea and the cerebro-spinal fluid. There may be still some doubt, of course, whether this was the primary cause of the original lesion.

Differential Diagnosis.

All sixteen patients were admitted to hospital with the provisional diagnosis of laryngeal diphtheria. To those accustomed to dealing with this disease the difference is obvious. The dramatic suddenness of the onset, the absence of true laryngeal signs and the absence of membrane are important points.

The condition known as laryngo-tracheo-bronchitis may present some difficulty. In this disease, which is frequently staphylococcal in origin, the onset may be rapid; but the obstruction is subglottic and due to thick, tenacious mucus in the larynx and trachea. There is no swelling of the epiglottis. Cases of asthma should not present any trouble, but retropharyngeal abscess may be difficult to exclude until a complete examination is made.

Confirmation of the diagnosis of œdema of the supraglottic region can be made with certainty either by direct vision or by digital examination. The latter method is to be preferred as being more rapid and certain and less distressing to the patient.

Treatment.

Trachectomy was shown to be a life-saving operation in this condition. Intubation with O'Dwyer's tubes was attempted in a few early cases, but was never successful. It was performed with great difficulty owing to the narrowed glottic opening, and when it was accomplished the swollen epiglottis closed over the top of the tube. Indications for tracheotomy were obvious distress, prostration, pallor or cyanosis. So long as the colour remained good and the child appeared to have plenty of fight, tracheotomy was deferred. At the first sign of change of colour or development of signs of exhaustion, operation was performed without delay. The operation presented no special difficulty if carried out early, but if it was delayed until prostration had become severe, collapse was likely to occur during the operation. Speed was then essential. One patient died after operation as the result of an incompletely controlled hæmorrhage into the trachea. As a rule the post-operative management was not difficult. Unless the wound became infected there was little discharge from the tube. In this regard the nursing was much simpler than in laryngo-tracheo-bronchitis or laryngeal diphtheria, in which blockage of the tube is frequent. The average time for retention of the tube was five days; but in no case was it more than a week. The wound always healed readily.

Sulphonamides were given in every case, in the form of sulphathiazole, sulphadiazine or sulphamerazine. Penicillin was also used in some cases, but as the probable organism is penicillin-resistant, it is doubtful whether this measure played any useful part in the treatment.

All patients were treated in a steam tent before and after operation, and oxygen was used freely.

Most patients developed some degree of anæmia before recovery was complete; but transfusion was necessary in only one case. This was in the case of the child who developed meningitis due to Friendländer's bacillus, an infection which was controlled only after two prolonged courses of sulphadiazine.

Hæmophilus influenzæ rabbit antiserum was used only in the three cases in which meningitis developed. It was undoubtedly a factor in the patients' subsequent recovery. Whether its use early in the disease would have accelerated the recovery of the other patients or prevented the onset of meningitis is still open to doubt.

Summary.

Sixteen patients were admitted to the hospital over a period of eighteen months with a condition of œdema of the supraglottic region particularly affecting the epiglottis and the aryepiglottic folds. The clinical picture was one of severe prostration and rapidly developing respiratory obstruction caused by partial occlusion of the glottic opening. There were two deaths, one of which occurred in the ambulance before the patient's admission to hospital.

In nine cases tracheotomy was necessary. This is considered to be a life-saving operation in this condition, and the importance of its early performance is stressed.

The role of *Hæmophilus influenzæ* as the causative organism is discussed.

Purulent meningitis is a possible complication of the condition.

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THE STUDY OF THE HUMAN BODY AFTER DEATH.[1]

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"MEN fear death as children fear to go in the dark", said Francis Bacon, and added: "It is as natural to die as to be born."

Throughout history study of the human body after death has been hampered by superstition and religious prejudice. This study has two main purposes: (i) anatomical, (ii) pathological. Knowledge of the normal anatomical structure of the body is an essential background for medical practice; knowledge of alterations wrought by disease is essential not only to establish the cause of death, but also for an understanding of the processes of disease in general.

Chronologically considerations of anatomy preceded those of pathology. In the time of Hippocrates (460 to 370 B.C.), anatomical knowledge was mainly gained from the dismemberment of animals slaughtered for sacrifices, and Aristotle (384 to 322 B.C.), a pupil of Plato, taught anatomy by the dissection of animals.

Dissection of the human body was first conducted on a fairy large scale at Alexandria in about 300 B.C. Three factors helped to establish anatomy as an independent science in Alexandria—namely, the practice of embalming the body, a lessening of religious prejudice, and the encouragement of the Ptolemies.

Galen (131 to 201 A.D.) followed the traditions of the Alexandrians, but he had not their opportunities of dissecting the human body. A long period of darkness followed Galen, and though there were occasional flashes of light, it was not until the time of Vesalius (1514 to 1564) that anatomy in the modern sense came to be revealed. Vesalius had difficulty in obtaining bodies for study, and it has been said that he secured his first human skeleton by stealing the remains of a hanged and roasted thief, outside the walls of Louvain (Ball).

When today you cross the Arno by the Ponte Vecchio and enter the heart of Florence, where the "David" of Michelangelo (1475 to 1564) is on one side and the "Perseus" of Cellini (1500 to 1571) on the other, you feel that you are in the atmosphere of the famous Florentine artist anatomists. Leonardo da Vinci (1452 to 1519) and Michelangelo did much of their dissecting in Florence and were among the artists who put new life into the study of anatomy.

Late in the sixteenth century, and at the commencement of the seventeenth, Fabricius (1537 to 1619) worked and taught at Padua, where Harvey was a pupil (1599 to 1603). During the eighteenth century an important factor in the advance of medicine was the presence in Great Britain of private schools of anatomy and surgery, especially that in Great Windmill Street, where William Hunter and John Hunter worked and taught anatomy, pathology and surgery. In the early nineteenth century the activities of the resurrectionists, especially in Edinburgh, became a matter of grave public concern, culminating in the murders committed by Burke and Hare. Burke at his trial was found guilty and addressed by the Lord Justice-Clerk as follows:

> Your sentence shall be put into execution in the usual way, but accompanied with the statutory attendant of the punishment of the crime of murder, *viz.*, that your body should be publicly dissected and anatomized.

In pursuance of the sentence of the court, Alexander Monro Tertius publicly dissected the body, and the following day not less than 25,000 persons viewed the remains of the murderer (Ball). In 1832 the *Anatomy Act* was passed, and body-snatching became a thing of the past. The act repealed the direction that the bodies of murderers should be dissected (Goodman).

An interesting historical review relating to "the supply of bodies for dissection" is presented by Goodman in the Arris and Gale Lecture (1944). Goodman discusses recent trends, the present position and the prospects for the future in Great Britain. In regard to the causes of diminution in supply he makes the following statement:

> The long-term cause which has been operating with gradually increasing force over the past 100 years is, of course, the improvement in the social and financial position of the poorest classes. Improvements in real wages, social legislation, and philanthropic efforts have all played their part, and there is little reason to expect that this process will not continue—indeed, certain aspects of social legislation already adopted in principle by the Government may be expected to have most important effects on supplies.

The same holds true for Australia, and unless some radical change is effected, the present lamentable situation with its inevitable lowering in standards of medical knowledge will become even worse than it is now. In my opinion the way to meet the situation is by educating public opinion along the right lines. I have great confidence in the commonsense and fairness of the man and woman in the street, and if they have the right example to follow I am sure they can be relied on to follow it. These people should be encouraged, by example as well as by precept, to leave written statements (accompanied by written consent of next-of-kin) expressing willingness for their bodies to be studied after death either by pathological post-mortem examination or by anatomical dissection. The example should be set by medical men, who are familiar with the problem, and who know precisely what is involved.

It was not until the time of Morgagni (1682 to 1771) that the human body was seriously studied after death from the point of view of pathology. The standard textbook prior to Morgagni's "*De Sedibus et Causis Morborum*" was Bonet's "*Sepulchretum Anatomicum*", which included a large collection of post-mortem reports from the sixteenth and seventeenth centuries. Morgagni's great book published in 1761 correlated clinical signs with pathological changes in the organs. Bichat (1771 to 1802), who laid the foundation of pathological histology, stressed the importance of tissues as contrasted with organs in the study of lesions in the body. As Long puts it: "Bichat formed the connecting link between Morgagni, who had so greatly influenced the teaching of organ pathology, and Virchow, who developed the concept of a pathology of cells." Facilities for the study of the human body from the point of view of pathology have greatly improved during recent years.

In the eighteenth century Morgagni worked in the Anatomical Theatre of Fabricius at Padua, which was built in 1594, and except for minor alterations remains unchanged to the present day. In this theatre there is a small area on the floor for the autopsy table, and six tiers completely surround the room. These tiers extend from floor almost to ceiling and are so narrow that onlookers must stand.

In the first half of the nineteenth century Rokitansky, working in Vienna, was an outstanding pathologist. Nine years ago the lecture theatre in Vienna, which seemed very old, had the autopsy table in front of the seats, which provided sitting accommodation for large numbers; those sitting at the back, however, were at a great disadvantage,

[1] Read at the annual meeting of the Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association on December 6, 1945.

because they were very little higher than those in front, and were too far from the body. In some American hospitals, about fifteen years ago, movable structures made of iron piping were wheeled around the autopsy table to form three sides of a square. This arrangement provided sitting accommodation for considerable numbers of students and gave a good view of the autopsy table, but the "seats" were not very comfortable.

A few years ago Lord Nuffield made a gift to the Royal Prince Alfred Hospital, Sydney, and this money, with more added, was used in building and equipping the Lord Nuffield Theatre of Pathology in the basement of the new Medical School of the University of Sydney. The amphitheatre is arranged to provide comfortable sitting accommodation for about eighty students, which corresponds to half the total number of students in an average year. A special system of lighting has been installed (in addition to that over the table), and when specimens removed from the body are held up to view under a bright light, the students in the back row are only about eight feet from them. Facilities for cutting immediate frozen sections are provided, and students participate in performing the examination of the body. Cooling of the cabinet in which the bodies are kept is adjusted to prevent decomposition but not to destroy the cellular structure of the tissues.

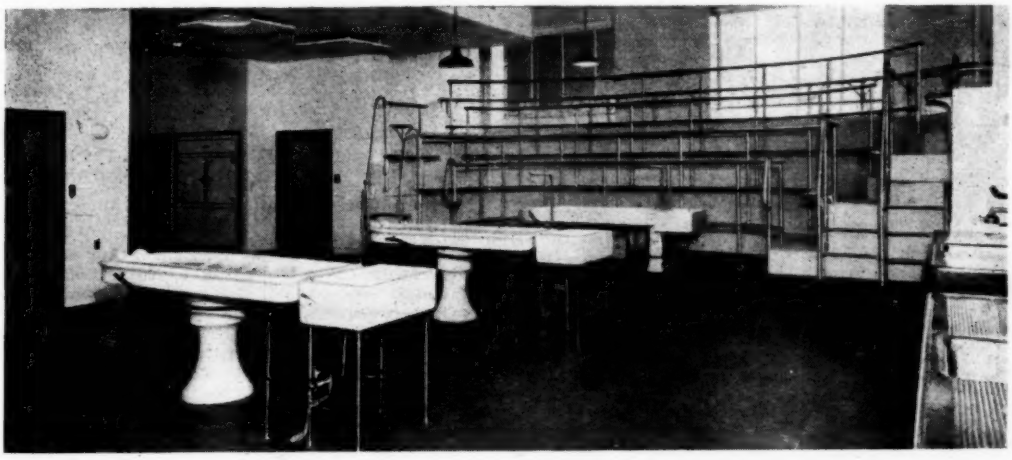


FIGURE I.

Lord Nuffield Theatre of Pathology (main theatre). The special system of illumination in front of the amphitheatre had not been installed when this photograph was taken.

A small chapel is in communication with the main theatre; in one end of this chapel is a viewing chamber, where the body is placed on a bier behind a large plate-glass window for inspection by friends of the deceased; a velvet curtain covers this window when the viewing chamber is not in use. Every endeavour has been made to show consideration for the relatives, and to make it clear to students that the term "dead-house" is obsolete and has been replaced by "theatre of pathology", a science laboratory where an endeavour is made to unravel the mysteries of disease.

Great advances have been made during recent years in diagnosis, but it still remains true that at a few autopsies the clinical diagnosis is shown to be completely wrong, and at a rather larger number partly wrong; even when the clinical diagnosis is confirmed, important abnormalities are often found which were unsuspected during life. Nearly a century and a half ago Bichat said:

> You may take notes, for twenty years, from morning to night at the bedside of the sick, upon the diseases of the heart, the lungs, the gastric viscera, etc., and all will be to you only a confusion of symptoms, which, not being united in one point, will necessarily present only a train of incoherent phenomena. Open a few bodies, this obscurity will soon disappear.

The same advice, in the main, holds good at the present day, and yet in our large metropolitan hospitals it is not unusual for a medical officer to fail to see the post-mortem examination of the body of a patient who dies while under his care, and a radiologist rarely seeks confirmation of a doubtful diagnosis in the autopsy room. For country practitioners the opportunity of checking clinical diagnosis by post-mortem examination seldom arises.

When a doctor does not ascertain what lesions were present in the body of one of his patients who dies, such a doctor may remain ignorant of the fact that some of the opinions he formed from clinical observation were entirely erroneous. This calls to mind the occasion referred to by Plato in "The Apology for Socrates", when Socrates approached a man who seemed wise to not a few but particularly to himself, and, after questioning him, went away saying: "This man imagines he knows, without really knowing. I, knowing nothing, do not even suppose that I know. On this point, at any rate, I appear to be a little wiser than he."

It used to be the custom to have a motto over the entrance portal to the post-mortem room in a hospital. At the Royal Prince Alfred Hospital the motto was "*In caelo quies*", and at the Sydney Hospital "*Etiam mortui prosumus*". It is true that even though dead we are of use, and indeed that is the theme of the present address; nevertheless it was thought that the public display of this motto might offend the sensibilities of some people, and it was not painted over the entrance to the new autopsy room in the Kanematsu Institute at the Sydney Hospital.

There appears recently to have been an awakening of interest on the part of some of our younger physicians in the study of diseased organs and tissues after death, and this is a hopeful sign for the future. I understand that it is proposed to establish more departments of pathology in country centres in this state, and these will prove a boon to country practitioners in their service to the people. The opinion has been expressed that the pathologists in these country centres should not be required to do work which calls for a knowledge of pathological histology, because of the need for long experience to gain confidence in this field of study. I do not concur in this opinion; no autopsy is complete without histological study of some organs and tissues, and sometimes the diagnosis cannot be determined until microscopic evidence is available, and even then occasionally remains uncertain. The performance of autopsies in large country centres is most desirable, as it is an important step in the direction of instituting clinical pathological meetings among groups of practitioners who are far removed from the metropolis.

While the encouragement of autopsies in widely separated centres is advocated, it must be recognized that some centralization of these activities is necessary in order that those who do the work may have enough practice to gain the requisite experience. An autopsy that is badly done might be better left undone. This calls to mind a request which came to the Department of Pathology of the University recently, that some attendants from a mental hospital should visit the Lord Nuffield Theatre of Pathology to pick up the essentials of post-mortem technique; I understand that a visit on one afternoon was contemplated. This request is reminiscent of the story which has been attributed to an eminent British bacteriologist. It is said that a young doctor called at this bacteriologist's laboratory to learn bacteriological technique while the wife of the young man waited in his motor car down in the street.

Bodies and museum specimens should be strictly anonymous so far as students are concerned; this rule is observed in all medical institutions of repute. Mixed medical museums are a mistake. Medical museums should be either for laymen or for scientists; those for scientists should not be open to laymen.

In the mental hospitals of New South Wales there are some 12,000 patients. In the larger institutions more laboratory facilities should be provided, and the number of autopsies performed should be greatly increased. The standard of service rendered by medical officers attached to mental hospitals would be raised considerably if they were given facilities for more extensive scientific investigation of the diseases from which their patients suffer.

Reports of Cases.

VOLKMANN'S ISCHÆMIC CONTRACTURE OF THE FOREARM.

By LEONARD BALL, F.R.C.S., F.R.A.C.S.,
Melbourne.

ISCHÆMIC contracture has long been looked upon as a sequel to tight bandaging or splinting, resulting in venous obstruction, although Volkmann himself, writing in 1881, considered it due to "a continuous stoppage of arterial blood".[1] A tight plaster cast may cause arterial obstruction, and further theories as to the production of ischæmic contracture are as follows: (i) contusion of a main artery, (ii) traumatic arterial spasm, (iii) arterial thrombosis or embolism, (iv) traumatic aneurysm or arteriovenous aneurysm. The condition most commonly follows supracondylar fractures of the humerus, when the brachial artery may be injured or stretched across the lower end of the humerus.

The following case, occurring in a young lad suffering from a fracture of his olecranon without displacement, who had no treatment prior to the onset of his ischæmia, is of interest from both the medico-legal and the clinical aspects.

Clinical Record.

J.M.O'N., aged seventeen years, fell from a tram on to his right elbow at about midnight on June 4, 1944. His arm was painful, but not unduly so, and he slept fitfully through the night; but in the morning he had considerable pain in the forearm and was admitted to the Alfred Hospital at about 11 a.m.

On examination the forearm was seen to be tense, swollen and painful, and appeared fluctuant; complete anæsthesia was present from just above the wrist joint to the fingers. The swelling of the forearm did not involve the hand. There was complete paralysis of the muscles of the forearm and hand, and the radial and ulnar pulses were palpable and of good volume. On June 4, after consultation with Mr. Balcombe Quick and Mr. Fay Maclure, it was decided to open the forearm, in the hope that a hæmatoma might be present which could be evacuated.

At operation an incision was made along the entire length of the anterior surface of the forearm and the deep fascia was incised, considerable bulging of the muscles being produced. The brachio-radialis appeared normal and was retracted to the radial side. The *musculus flexor digitorum sublimis* (humeral head) appeared normal

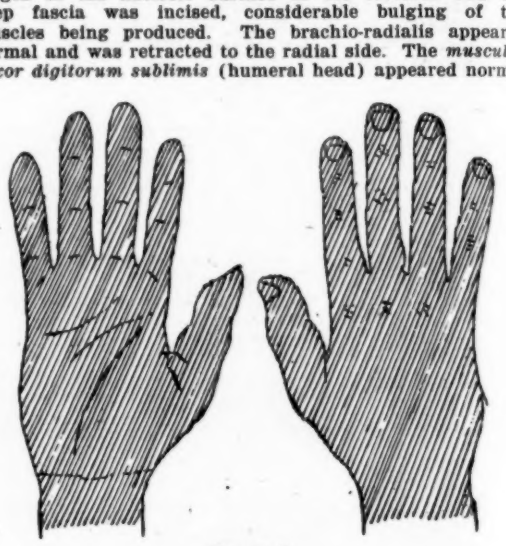


FIGURE I.
Showing level of anæsthesia on the patient's admission to hospital on June 5, 1944.

and was retracted to the ulnar side; thus the radial head of origin of the muscle was exposed. This latter was paler than normal and did not bleed easily, nor did it contract readily on stimulation. The muscle fibres were split and the *musculus flexor digitorum profundus* was exposed; this appeared normal, except that it was infiltrated with blood, presumably from the fractured

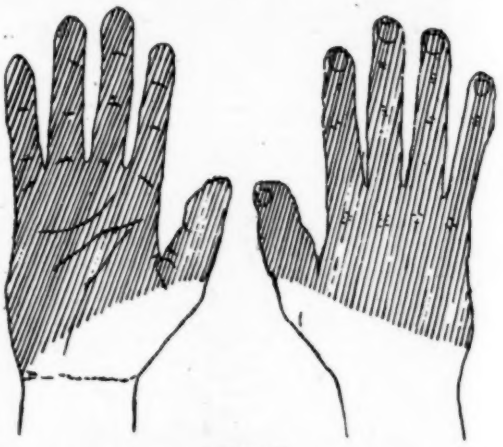


FIGURE II.
Showing level of anæsthesia on September 1, 1944.

olecranon. No attempt was made to suture the incision, and indeed, on account of the tension of the forearm, it would have been impossible to do so. The wound was dressed with "Vaseline" gauze.

On his return to the ward the patient's arm was kept elevated in a sling suspended from a framework above the bed, this being the position of greatest comfort for him. There was no evidence of post-operative infection, and on June 12 (one week after operation) the wound was

dressed, when it was found that the swelling was considerably reduced and some passive movement of the wrist joint was possible. On June 19, at the next dressing, the swelling of the forearm had subsided still further, but there was considerable evidence of trophic changes in the fingers, especially on the dorsum of the proximal interphalangeal joints, where blisters were present. Complete anæsthesia was still found to exist.

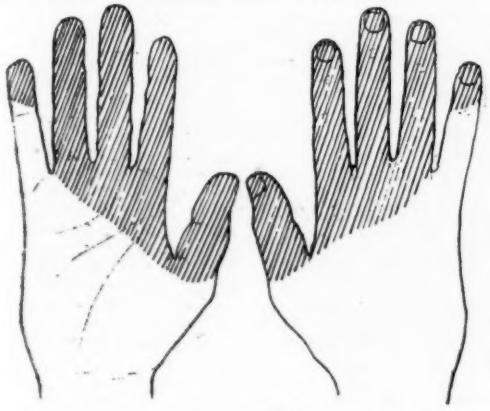


FIGURE III.
Showing level of anæsthesia on February 1, 1945.

The wound of the forearm healed without incident, and the hand and forearm gradually assumed the position typical of a Volkmann's ischæmic contracture. Attempts were made to prevent this deformity by splinting, but these had to be abandoned owing to the trophic changes in the fingers and hand, the slightest pressure causing blisters. The area of anæsthesia gradually became smaller, as is shown in Figures I, II, III and IV.

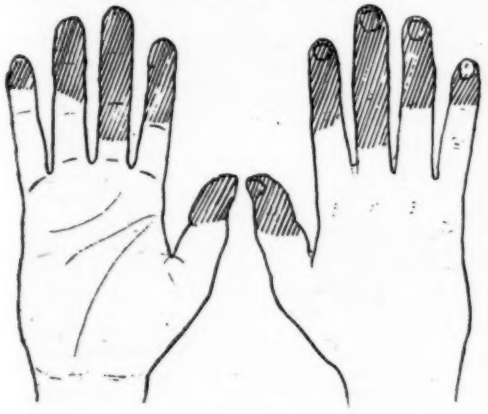


FIGURE IV.
Showing level of anæsthesia in July, 1945.

Although no special attention was directed to the fracture of the olecranon—in fact, the forearm was flexed on the arm during the period of treatment—the fracture united perfectly without any disability of the elbow joint.

Comment.

Although this patient developed a typical Volkmann's contracture (as shown in Figure VI), yet at operation the ischæmic process seemed to be limited to the radial head of his *musculus flexor digitorum sublimis*. However, other structures were involved, as was shown by the complete paralysis of his forearm and fingers and the total anæsthesia distal to his wrist. The anæsthesia slowly disappeared, and also presumably the motor nerve supply to the forearm muscles improved. The result may have been improved by an excision of this muscle, or perhaps a detachment of its origin at the time of the original operation.

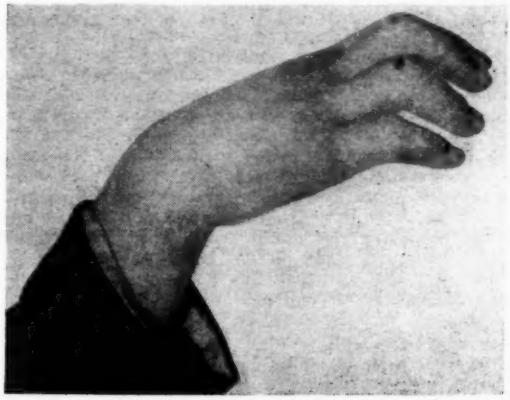


FIGURE V.
Photograph taken in September, 1944, showing developing ischæmic contracture and trophic changes.

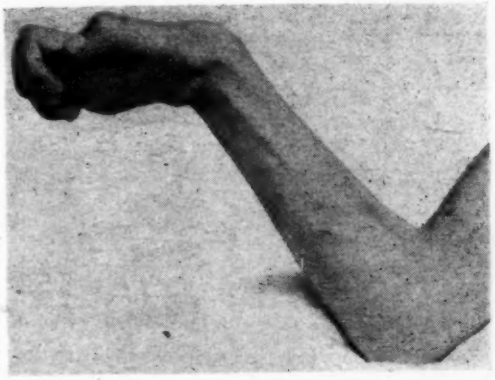


FIGURE VI.
Photograph taken in June, 1945, showing fully developed Volkmann's contracture.

It is fortunate for the doctor concerned that he did not examine the patient earlier, as it is probable that any treatment directed toward the fractured olecranon would have been blamed, or at least been considered a contributory factor, in the development of his ischæmic contracture.

Reference.

(1) R. Watson-Jones: "Fractures and Injuries", Third Edition, Volume I, page 121.

UNKNOWN FOREIGN BODIES IN THE LUNG.

By J. B. CLELAND,
Marks Professor of Pathology, University of Adelaide.

A WOMAN, aged thirty-three years, suffering from schizophrenia, died at a mental hospital at 7.30 p.m. on July 23, 1944. She had always been thin. She had suddenly

become ill on the day on which she died, suffering from dyspnœa, a flushed face and physical signs of a pneumonic state. In the lower and anterior part of the upper lobe of the right lung was a red, consolidated area, one and a half inches (3·7 centimetres) in diameter, with pale specks in it, possibly minute abscesses. In the lower lobe of the right lung a superficial dark hæmorrhagic-looking patch was seen.

Microscopic examination of the affected lung revealed considerable consolidation, the alveoli being filled with coagulated exudate, red cells or polymorphonuclear cells, or in some places loosely with macrophages. Embedded here and there in the consolidated areas, and usually in groups, were a number of foreign bodies. These were oval in shape and varied in size (some measurements being 52μ—evidently cross section) from 83μ by 66μ to 116μ by 83μ. There appeared to be a thin capsule which stained a reddish colour with hæmatoxylin and eosin, and the contents seemed divided into about 10 or 12 spaces by reddish, irregular septa giving a vague honeycomb or reticular appearance. Sometimes these spaces looked as though they were independent oval structures. In one case the contents, which seemed to comprise four irregular elongated bodies, had shrunk, leaving apparently a capsule behind. With iodine a faint blue tint appeared. The bodies were quite unlike pollen grains. Professor T. Harvey Johnston did not recognize them as of animal origin (for example, ova) and Professor J. G. Wood considered that they were not vegetable in nature. Can anyone suggest what these bodies are from this description and the sepia wash drawing (Figure I)?

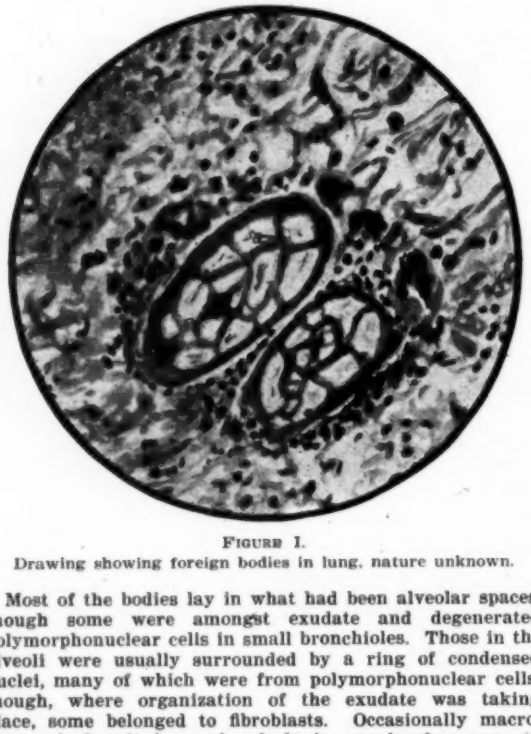


FIGURE I.
Drawing showing foreign bodies in lung, nature unknown.

Most of the bodies lay in what had been alveolar spaces, though some were amongst exudate and degenerated polymorphonuclear cells in small bronchioles. Those in the alveoli were usually surrounded by a ring of condensed nuclei, many of which were from polymorphonuclear cells, though, where organization of the exudate was taking place, some belonged to fibroblasts. Occasionally macrophages had united round a body in an alveolar space to form small foreign-body giant cells. The bodies were irregularly distributed in the sections, and in one low-power field 12 were counted; but in considerable areas of consolidated lung none were found. If these bodies were not responsible for the condition in the lungs as a whole, they certainly played an important subsidiary part.

TETANUS TREATED WITH PENICILLIN; RECOVERY.

By JOHN L. GROVE,
Launceston, Tasmania.

Clinical Record.

THE patient, a male, aged twenty-four years, had been healthy except for chronic *otitis media*; this in 1940 spread to the left mastoid and gave rise to meningitis, which responded to sulphonamide therapy. He was perfectly well until five days before his admission to hospital. He is a motor truck driver, and on that day noticed that any heavy lifting gave rise to pain low in the centre of the front of his chest. This was not severe, and he was able to carry on with his work. Four days before his admission to hospital the symptoms were unaltered, except that at lunch time he had one sudden, severe, cramp-like pain in the chest, of short duration. He still worked and slept well that night. He suffered from no general malaise and no stiffness anywhere. Three days before his admission to hospital he was having fairly frequent attacks of cramp in the chest and abdomen, and was unable to work. He remained in bed and called a medical practitioner, who could detect no abnormality and considered the condition dyspeptic. That night he had several spasms of increasing severity and frequency, and these continued for the next two days and nights. By the morning of his admission to hospital, on May 13, 1944, he had become exhausted and called another medical practitioner, who found him gravely ill. He was admitted to a private hospital with a provisional diagnosis of acute appendicitis, and was examined by me at midday, by which time the diagnosis had become obvious.

On examination, the patient was sweating profusely and had a furred tongue and most offensive breath. Board-like rigidity of the abdomen was present, and any attempt at palpation brought on typical generalized tetanic spasms. His temperature was 101·6° F., his pulse rate was 124 per minute, and his respirations numbered 28 per minute. There was no wound except a small puncture on the right hand.

A diagnosis of tetanus was made and confirmed by a surgeon called in consultation in view of the provisional diagnosis mentioned above. Treatment with penicillin and serum was commenced immediately. Penicillin was given intramuscularly in doses of 20,000 units every three hours. The patient complained that the penicillin injections were painful, until the site was changed from the buttock and thigh to the deltoid, where the injections seemed to be quite painless. (This site, moreover, involves minimum disturbance of the patient.) Penicillin treatment was continued for ten days with a 25% reduction in dosage on the fifth day, a total of 1,710,000 units being used. Serum was given both intravenously and intramuscularly for the first three days, and thereafter intramuscularly only. Serum therapy was continued for twelve days, a total of 270,000 units being given. The nursing staff expressed the opinion that a definite improvement seemed to follow each dose of serum.

A variety of sedatives was used. "Avertin" was the sheet anchor. Twelve doses were given during the first nine days in hospital, the total used being 62·1 mils. At first the "Avertin" gave from six to eight hours' sleep with freedom from spasms, the period of relief gradually diminishing to about four hours. The only disadvantage of "Avertin" seems to be that while the patient is under its influence, it is difficult to keep up fluid intake. This had been noted in previous cases in which "Avertin" was used. In order to keep up fluid intake, it was necessary to give for periods between doses of "Avertin" a drug which gave a measure of control of spasms with at least reasonable freedom from pain, and yet allowed cooperation from the patient. Morphine filled this need well and gave rise to no anxiety. A total of four and five-twelfth grains was given. Morphine was on occasion combined with hyoscine, of which a total of one-tenth of a grain was given. "Nembutal" was tried on three occasions, but gave no

relief. "Sodium Amytal" was found useful after the fifth day and toward the end of the illness, when spasms were decreasing in severity and number, it was used in place of "Avertin"; 87 grains of this drug were used in all. "Sodium Amytal" seems to have a place in the treatment of tetanus, but does not appear to be as useful as "Avertin" in the severe stages.

Spasms, which were precipitated by the slightest stimulus—for example, a motor car passing down the street outside the hospital—continued until the thirteenth day in hospital; 252 spasms were recorded. This is a conservative estimate, as no record was kept on the first day, and for some reason the staff did not usually record spasms occurring as the result of the naturally frequent attention to the patient. Abdominal rigidity persisted till the sixteenth day. The jaw stiffness persisted till after the patient's discharge from hospital on the nineteenth day. Even on the seventeenth day he could open his mouth only half to two-thirds of the normal extent.

His temperature ranged between 99° and 101·6° F. for the first six days and then gradually fell, remaining normal after the sixteenth day in hospital. The pulse rate, apart from that recorded during spasms, did not rise above 120 per minute, and it gradually settled with the temperature. The respiration rate was 44 per minute on the third day in hospital, and this also gradually fell with the temperature.

The urinary output for the first twelve days totalled 303 ounces, whereas the fluid intake was 819 ounces. The tremendous loss of fluid mainly by perspiration is one of the features of severe tetanus. In this case there was a loss of over three gallons of fluid in twelve days (not counting the actual loss of weight of ten pounds during the illness), and this bears out the need for strict attention to intake if dehydration is to be avoided. Diet was fluid until the fourteenth day (the last spasm occurred on the thirteenth day), when semi-solids were started. He was able to eat meat on the seventeenth day despite some residual stiffness of the jaw.

The bowels, which had last acted two days before his admission to hospital, remained unopened until the ninth day in hospital. Enemata were given twice without success and on each occasion seemed to make his condition temporarily worse. Finally the bowels opened into the bed in response to the last dose of "Avertin".

Comment.

While no conclusions can be drawn from one case, it was thought that an analysis of the records of previous patients treated in Launceston might be of interest. Records of 16 other patients since 1930 were traced; of these, seven recovered. The ages ranged from two to seventy-three years. The incubation period (which could not be determined in five cases) averaged five days in the fatal cases and eleven days in the others. The period of onset (that is, the period between the first symptom and the first spasm, which was not known in three cases) averaged 2·4 days in fatal cases and 2·5 in others.

Only two of the other patients who recovered could be classed with this patient as severely affected. In one of these cases the incubation period was 18 days, and although 388 spasms were recorded over eleven days, the patient's condition never gave rise to anxiety, his temperature being normal by the fourth day and remaining so. In the other case, in which the incubation period was unknown, the patient had spasms for six days and the initial temperature of 98·8° F. returned to normal after the second day in hospital.

It would seem fair to state that the case described is probably the most severe case of the series in which recovery occurred. As handicap additional to the severity of the disease, the patient was nursed in a private hospital by a staff none of whom had previously nursed a patient suffering from tetanus, and unfortunately in a noisy room. The patient had a rapid convalescence and has remained at his work since, apparently none the worse for his illness.

Reviews.

DERMATOLOGY.

AFTER a period of seven years, Dr. E. H. Molesworth has presented a second edition of "An Introduction to Dermatology".[1] The first edition was reviewed at some length in this journal on April 10, 1937. Despite the difficulties associated with the war, Molesworth has found sufficient time and energy to produce a volume which is distinctly better than its predecessor. There has been some rearrangement in the subject matter. The chapters have been divided into seventeen instead of thirteen, and the number of illustrations has been increased by eleven. Molesworth admits the collaboration and influence of several overseas authorities, perhaps the best known being Dr. Frederick Goldschlag, late of Poland. In addition, Dr. R. Kaye Scott, of Melbourne, has brought the section on radium right up to date. However, as in the first edition, it is the pen of the experienced and dogmatic Molesworth which dominates, and this is as it should be.

Many dermatological authors include so many treatments (without indicating their personal preference) that an inexperienced practitioner may be at a loss to know just what to prescribe. This is far from the case with the author under review. His book is easy to read and the treatments are clear-cut.

Molesworth makes no claim that his textbook is complete. There are certain omissions, some of which are certain to be included in the next edition. It will now be convenient to enumerate some of them. (i) There is no reference to penicillin. This is, of course, explained by the fact that the book was in the press before this great therapeutic agent was available. (ii) As a textbook for students there should be one or two illustrations in a chapter dealing with the anatomy of the skin. (iii) There should be a special chapter devoted to drug eruptions. Certain eruptions due to the absorption of drugs are described here and there in the text, but the book would be far more complete with this inclusion. (iv) There is no reference to Thorium X, which has recently been advised for port-wine stains and tinea of the nails. (v) "Mouse plague" has been omitted from an excellent article devoted to fungous infection. Outbreaks of this form of tinea are not uncommon in certain of the wheat areas in Australia. (vi) Very little has been written concerning skin diseases under tropical war conditions. There is no description of a fairly common condition known by such names as tropical lichenoid dermatitis, New Guinea *lichen planus* or a *lichen-planus*-like eruption. Experience in tropical war conditions was only gathering when Molesworth completed this work.

It is not proposed to deal *seriatim* with every chapter; they are all consistently good, but the outstanding ones deal with the innocent and malignant tumours of the skin. Molesworth from his vast experience is at his best in dealing with the X-ray treatment of both rashes and tumours. Probably this aspect of his book will make the greatest appeal to the dermatologist and radiotherapist. For the student and general practitioner the interest may be only academic. Here is a definite technique laid down. There should be no misunderstanding in the interpretation of any dosage of X-ray therapy for a particular eruption or neoplasm of the skin. There are a few statements that need commenting on. (i) On pages 50 and 51, Molesworth has laid stress on the importance of avoiding chemical irritants in the treatment of simple skin diseases. He especially condemns the sulphonamides, and surely all experienced dermatologists are in agreement. A competent medical officer recently returned from the tropical areas stated that the commonest causes of dermatitis in these zones were sulphanilamide and acriflavine. If it did not spoil the appearance of the book, one would like to see a special chapter or article in big print condemning the indiscriminate prescribing of external irritants. (ii) X-ray treatment of eczema is discussed on page 55, and the following technique for obstinate cases is advised: 110 kilovolts, a filter of one millimetre of aluminium and a dose of 250*r*, the treatment to be repeated once a week for three applications. For the lichenoid or infiltrated type there may be no objection, but for the acute or subacute forms of eczema most authorities prescribe much more con-

[1] "An Introduction to Dermatology, with a Chapter on the Theory and Technique of X-Ray and Radium Therapy", by E. H. Molesworth, M.D., Ch.M. (Sydney), F.R.A.C.P., Dip. Rad. (Sydney), with a Foreword to the First Edition by Professor Josef Jadassohn; Second Edition; 1944. Sydney: Angus and Robertson, Limited. 8¼" × 5¼", pp. 592, with 162 illustrations in the text. Price: 37s. 6d.

servative doses. Molesworth may do likewise, but he does not make any mention of smaller doses. (iii) The incidence of *lupus vulgaris* in Australia is discussed on page 136, and the author states that he has seen only one person in Australia who developed this condition. Dermatologists in other States than New South Wales have seen occasional cases in subjects who have never been out of Australia, but the main point is that *lupus vulgaris* is a rare condition in this country. (iv) Thallium acetate epilation (page 210) is still recommended in certain circumstances for *tinea capitis*. Many now consider that thallium acetate should no longer play a part in the treatment of ringworm of the scalp, and the Ministry of Education of England does not sanction its use (*British Medical Journal*, September 15, 1945, page 348). Molesworth has given detailed instructions which have been successful in several hundred cases. (v) Iontophoresis (page 215) is favoured for the treatment of otherwise resistant ringworm conditions between the toes. This is a new form of therapy. It would be interesting to know the incidence of recurrences under hot conditions. (vi) X-ray treatment for certain types of *lupus erythematosus* is advocated (page 402). It has been handed down or taught for years that X-ray treatment is contra-indicated in this condition. It is thought that its application may produce skin cancer. Molesworth is emphatic that in the dosage he employs this complication has never occurred. (vii) X-ray treatment of vascular nævi is mentioned on page 433. The author has for years used this method, and to him largely belongs the credit for its present popularity in Australia. With the introduction of the new shock-proof X-ray plants, this treatment is simplified. What took hours or days to do with radium may be achieved in a matter of minutes.

The book ends with a notable chapter by Dr. R. Kaye Scott, entitled "Technical Aspects of Radium Therapy in Dermatology". It is probable that no better or more concise article has ever been written. Unfortunately it is a fact that certain practitioners with little or no training in radiotherapy possess radium plates. Their dosage and results are often uneven, especially when they treat early squamous-celled carcinoma. Scott has clearly explained how the dosage of plates tends to be greater in the centre and weaker at the margin. Those who dabble with radium are exhorted to study this chapter.

In conclusion, this textbook can be confidently recommended to students, practitioners and dermatologists. For the last-mentioned it is especially valuable for its presentation of the subject of radiotherapy. This volume is a credit to Australian dermatology and is worthy of a place in any medical library in the world. More than a word of praise is due to the publishers, for the paper, print, binding and illustrations are of high order.

SURGERY OF THE RECTUM.

GABRIEL'S excellent work, "The Principles and Practice of Rectal Surgery", now appears in a third edition.[1] As stated in our reviews of earlier editions, the work is of considerable merit. The reader is taken along quietly, a start being made with an interesting, instructive and elaborate chapter on the surgical anatomy. The diagnosis of rectal conditions and the general principles of rectal surgery are then discussed. Thus the simplest of rectal diseases and their symptomatology and treatment are dealt with in turn, until finally, one of the most difficult of all surgical abdominal conditions is elaborated upon, namely, malignant disease of the rectum. In giving a separate chapter to malignant disease of the anus, the author appears to be splitting straws, but as the lymphatic spread of the anus is different from that of the rectum, the only comment that can be made is that such a diversion is in keeping with the thorough set-up of the book.

The diagrams dealing with the anatomy are masterly, and those of operative technique are easy to follow; it is interesting to learn that "old piles" give little response to injection. Apparently piles must be caught early for success with injection. It is questionable whether correction of habits will not effect a cure in many early cases of piles without recourse to injection, much less to surgery, being necessary.

A most interesting chapter appears dealing with rectal prolapse. It is surely very rare to see prolapse so extensive as to necessitate partial colectomy. In such cases Roscoe Graham considers that the fault lies in the anterior rectal wall. Surgeon Rear-Admiral Gordon Taylor is quoted on page 236 in the discussion on rectal injuries in war.

Some Australian surgeons serving with the Australian Imperial Force in the Middle East think that colostomy is a wise step always in anticipation of trouble after rectal injury. About a hundred pages are devoted to carcinoma of the rectum and every page is interesting. Some years ago an American writer said, "No surgeon can discuss intelligently cancer of the rectum without quoting Ernest Miles at every page", yet we find no description of his work, only a mere reference where to find it. It is surely not good surgical technique to turn into the abdomen a structure from a dirty area like the anus.

If perineo-abdominal excision is carried out in one stage, there will be times when the growth is so enormous that an equally large opening will have to be made in the iliac region to bring the mass out to establish a left inguinal colostomy. In any case it appears a laborious procedure to open the abdomen for inspection and then, when operation is approved, to start operating at the perineum, and when finished there to return to the abdomen for completion of the operation. Preliminary colostomy is often a necessity. Many a patient with a supposedly inoperable tumour improves considerably after a colostomy. Even patients with cardiac failure have been made fit for operation after a preliminary colostomy and have lived for years after radical operation. In obstructive types of growth, when effective lavage is not possible, preliminary colostomy is indicated. It is noted that in about 40% of the author's cases, when all ages are taken into consideration, tumours are inoperable. Lower percentages are claimed by some Australian surgeons and preliminary colostomy in doubtful cases has much to do with the lower figures. If patients are carefully selected for radical operation, there need be no mortality rate, and with the advent of the sulphonamide drugs the fear from sepsis is lessened considerably. In previous criticism of this really excellent work the advocacy of perineo-abdominal excision of the rectum for cancer as a routine measure has been questioned when so much can be said for the abdomino-perineal operation of Ernest Miles.

[1] "The Principles and Practice of Rectal Surgery", by William B. Gabriel, M.S. (London), F.R.C.S. (England); Third Edition; 1945. London: H. K. Lewis and Company Limited. 9¾" x 6", pp. 442, with many illustrations. Price: 45s. net.

DISEASES OF THE CHEST.

THE second edition of "Introduction to Diseases of the Chest", by James Maxwell,[1] will be welcomed by both teaching physicians and students, and although, as stated in the preface, the book is slightly smaller, the quality is still the same. The physical examination of the chest is described in a manner that would have pleased even Laennec, and the analysis of the symptomatology is noteworthy for its clarity. The importance of the habits, occupation and background of each individual patient in relation to the history of his disease is stressed.

Students especially will appreciate the chapter on radiology, in which the radiological appearances of the various chest conditions are discussed. The illustrations, most of them reproductions of skiagrams, are excellent, and printed as they are all together at the end of the volume, they enable an easy comparison of the various appearances to be made.

The many factors concerned in the causation of asthma are well discussed and the chapter on this subject contains all that students need to know. The pneumonias are dealt with fully, and the fact that forty-five pages are allotted to pulmonary tuberculosis is an indication that this important subject is treated adequately. The technique of collapse therapy is well described.

From an Australian viewpoint it is strange to find that hydatid disease of the lung is merely mentioned in a few lines and that no account of the treatment of this widespread disease is given. This would appear to be similar to finding little mention made of malaria in a textbook of medicine, published in any country just because there happened to be no Anopheles mosquitoes in that country. Even assuming that the disease is relatively uncommon in Great Britain, all students should know the special tests used in its diagnosis, and also the surgical technique used in its treatment.

Another surprising omission is to find no mention made of penicillin therapy, but this perhaps is explained by the fact that the preface is dated May, 1944, although the book was not published until eleven months later.

[1] "Introduction to Diseases of the Chest", by James Maxwell, M.D. (London), F.R.C.P. (London); Second Edition; 1945. London: Hodder and Stoughton Limited. 8½" x 5½", pp. 335, with many illustrations. Price: 12s. 6d. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 16, 1946.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

"PALUDRINE."

THE special article that appears on page 234 is one of the most important that has been published in this journal. It deals with the discovery and evaluation of "Paludrine", the new and potent antimalarial drug, and it shows how valuable have been the researches carried out at Cairns by the team of Australian workers of whom Brigadier N. Hamilton Fairley was director. The article should be read in conjunction with a leading article entitled "Triumph Against Malaria", which appeared in the *British Medical Journal* of November 10, 1945, of which every member of the British Medical Association in Australia should have received his copy. Though these two articles will give to Australian doctors the most up-to-date information on "Paludrine", there are several points in our special article on which stress should be laid.

In the first place it should be made quite clear that headquarters of the Australian Military Forces was in no way responsible first for its silence, and then for what must have appeared to some persons as an equivocation on its part, when it denied officially that "Paludrine" was being extensively investigated in Australia. The unfortunate double use of the name created a difficulty. In 1944 the name "Paludrine" was given by Imperial Chemical Industries, Limited, to a compound not so effective as the new drug and known in their laboratories as M.3349. When the British Press and radio announced the discovery of "Paludrine" (the newer and more effective agent with the laboratory name of M.4888, not the previously known M.3349), there was not sufficient time to notify the Australian Army authorities either that the name "Paludrine" had been given to M.4888 or that "security silence" in regard to it had been lifted. Later on, when Dr. D. G. Davey, the discoverer of "Paludrine" with F. H. S. Curd and F. L. Rose, was in Australia, a Press conference was called by the Director-General of Medical Services at Victoria Barracks, Melbourne, and a statement was given to Press representatives. With one or two exceptions the Press did not publish an accurate account of these statements. These happenings do not detract in any way from the value of the work that has been carried out at Cairns by the team of officers from the Australian Army Medical Corps. It is to the high value of this work that attention should next be directed. When Dr. D. G. Davey was in Melbourne he said that as a result of the work of the research unit at Cairns, "we have learnt in six months what might easily have taken years in the ordinary way". The work at Cairns has, in the words of our special article, "confirmed and extended in human malaria the results obtained by Dr. D. G. Davey and his team in bird malaria". The most important conclusion drawn from this work is that "Paludrine" in non-toxic dosage is a true causal prophylactic in malignant tertian malaria and a partial causal prophylactic in benign tertian malaria. In this it is superior to all known antimalarial drugs. Special mention should also be made of the difference, described as "considerable", which exists between the effective therapeutic dose and the toxic dose. The discovery of this "most potent antimalarial drug known" is indeed a triumph for British chemotherapy. We do well at the same time to reflect that what appeared in the early stages of the war with Japan to be a dire and irreparable disaster, provided an opportunity and a stimulus to research, for the results of which mankind will never cease to be grateful.

THE FEDERAL MEDICAL WAR RELIEF FUND.

FOR some considerable time before the fighting ceased in the recent war, the Federal Council of the British Medical Association in Australia had before it proposals to establish a Federal Medical War Relief Fund. The question was not whether a fund should be established, but when was the most suitable time for such an important action. That a war relief fund would find an enormous field for its activities and would be of untold benefit to incapacitated service medical officers and their dependants was clear from the experience gained with the Federal Medical War Relief Fund established in connexion with the war of 1914-1918. From May, 1919, when that fund began to be administered, until December 31, 1945, loans of varying amounts, totalling £23,752, had been made to 47 persons. As a matter of fact, loans are still being repaid by five beneficiaries who are entitled to this assistance. During the existence of the fund 32 persons have received from the fund gifts amounting in all to more than £10,066. At December 31, 1945, seven persons were receiving regular assistance from the fund, and these included widows, unmarried daughters and children of deceased medical officers whose education is not yet complete. To this record of achievement must be added the fact that the cost of administration of the fund from its inception has amounted to only £778 3s., an average of less than £30 a year. At December 31, 1945, the assets on hand amounted to £9,097 15s. 4d. It should perhaps be pointed out that this fund can be used only for beneficiaries affected by the war of 1914-1918. At its meeting in September, 1944, the Federal Council resolved to establish a Federal Medical War Relief Fund in connexion with the recent war and

set out the objects of the fund under the following six headings.

1. To assist members of the medical profession who have been disabled and the dependants of those who have died as a result of enemy action or of sickness contracted while serving in the armed forces.

2. To issue loans with or without interest to medical men who, as a result of enemy action, may require temporary financial assistance.

3. The fund be established by donations from members of the profession in all of the States, and to commence the fund an effort should be made in each State to secure at once as large initial donations as possible.

4. The fund to be controlled by the Federal Council.

5. A local committee of management to be appointed by the Branch Council in each State consisting of three members of the British Medical Association.

6. The Federal Council to have the power to use any surplus funds for the purpose of medical benevolence.

At its meeting in March, 1945, the Federal Council received the approval of the six Australian Branches of the Association and it proceeded to appoint as trustees of the fund Dr. George Bell, Dr. W. F. Simmons and Dr. G. C. Willcocks. It also decided that each Branch Council should be asked to appoint a local committee of management consisting of three of its members. The legal advisers of the Council were also to be consulted in regard to the drawing up of a deed of trust for the fund. The deed of trust was presented to the Council at its meeting in October, 1945, and adopted. Everything is thus ready for the launching of the appeal for donations to the members of the Branches.

In a matter such as this it should not be necessary to go into long explanations of why a fund such as the Federal Medical War Relief Fund should come into being and why every single member of the medical profession should give not merely, in the words of another appeal that is sometimes made, as his circumstances in life may fairly warrant, but in such a way that his gift will represent a personal sacrifice. There may possibly be some who would suggest that the State which accepted service from medical men and women should and will, in the event of their decease, accept the burden of caring for their dependants or of helping them with gifts or loans of money if their peculiar circumstances should call for such action. This is a specious kind of argument that will be made only by non-medical persons who cannot see things in their true perspective. The great majority of medical practitioners will not think along these lines. If large numbers of doctors could be credited with ideas of this kind the many schemes for the protection of practices would not have been so successful as they have been during the war and a spirit of comradeship would not have been so evident as it is today. Men and women who have served with the three branches of the armed forces have been named in these columns as being among the cream of our citizens. They have given freely of all that they had and many have given their lives. It is little to ask that we, the men and women who have come through this upheaval, some of us scatheless, some with scars of one kind and another, shall give aid to our own kith and kin, our own brothers in medicine, or to those dependent on them if they are in need. The State will do a certain amount, it is true, but no government, no statutory body that has to be bound by regulation and red tape, can give so personal, so sympathetic or so complete a kind of assistance as has been given by the Medical Relief Fund of the war of 1914-1918. The fund now to be established will do the same and more, and it covers the widest possible field—its provisions will apply to all medical practitioners and not merely to those who are members of the British Medical Association. The objective set is £50,000. Some will give large amounts; others will not be able to do this. But what is important is that every member shall give something. For those who from necessity or habit pay attention to these things, it may be mentioned that gifts to the fund will be subject to the concessional rebates of tax provided by Section 160 (2) (*g*) (iii) of the *Income Tax Assessment Act*, 1936-1945. A personal appeal is being addressed to every Branch member by the President of the Federal Council. In case the President's appeal should not be noted or interest should not be stimulated without delay, readers will help if in their chance meetings with their colleagues they become persistent publicity agents. Every member will do well to remember the old tag: *"Bis dat qui cito dat."*

Current Comment.

RETROPUBIC AND EXTRAVESICAL PROSTATECTOMY.

A RECENT report by Terence Millin on what he calls retropubic prostatectomy by a new extravesical technique[1] has received such glowing commendation from *The Lancet* that critical notice must be taken of it. In performing this operation, Millin uses low spinal anæsthesia with "Pentothal" or with "Pentothal" and "gas-oxygen". Cystourethroscopic examination is made to exclude unexpected pathological changes in the bladder and to study the configuration of the gland. After the bladder has been emptied an incision is made in the middle line, extending upwards from over the pubis for a distance of two and a half to three inches, depending on the obesity of the patient. The aponeurosis is incised in the length of the wound. The recti are separated in the middle line and the index finger sweeps upwards to the retropubic fat and the retroperitoneal fold, and a modified Harris self-retaining retractor is introduced. The veins running upwards on the anterior and lateral aspects of the prostate in the prevesical layer of the endopelvic fascia are underrun and ligated. The fat still adherent to the lateral aspects of the gland is gently sponged downwards and outwards. A medium-sized swab is pushed into each lateral recess. With a diathermy knife or number 15 Bard Parker a curved transverse incision, convex downwards, 1·0 centimetre distal to the bladder neck, is made through the prevesical fascia and true prostatic capsule. The lower flap is undermined towards the apex of the gland and the upper flap retracts owing to the pull of the severed pubo-vesical muscles. The false capsule is now exposed and an inverted-V incision is made down to the adenoma. The lateral lobes are freed from the false capsule and the dissection is continued with either forceps or finger. The lateral lobes, and when it is present the middle lobe, are freed digitally from below upwards until they present in the wound, being attached solely in the region of the bladder neck. The adenomatous mass is detached with scissors or diathermy knife. The edges of the capsule are secured with four pairs of Allis forceps. By traction on these and by suitable manipulation of flexible lamp and sucker the prostatic bed is carefully inspected. The two prostatic arteries, if not already secured, are coagulated,

[1] *The Lancet*, December 1, 1945.

and any other obvious bleeding vessels are similarly treated. A number 18 Harris catheter is passed along the urethra into the prostatic cavity and guided into the bladder. The defect in the false capsule is closed with a continuous catgut suture. The incision in the true capsule is closed with interrupted sutures. The packs are removed from the lateral recesses, the field of operation is swabbed dry and sprayed with sulphanilamide powder. The wound is closed, a small drain being left down to the suture line in the true capsule. Bilateral vasectomy is then performed. The catheter is irrigated with a solution of flavine to free it of any blood clot that may have collected during its passage through the prostatic bed. Four ounces of fluid are left in the bladder and the catheter is spigoted. The spigot is soon released and the tubing is placed in a Winchester bottle by the bedside. Only in an exceptional case has any post-operative syringing of the catheter been necessary. The catheter is removed on the sixth or seventh day; the patient is usually ready to leave hospital on the fourteenth day.

Millin's twenty operations have been performed for the following conditions: fibrous gland with sclerotic bladder neck, two cases; calculous prostate, one; pure lateral lobe hypertrophy, two; generalized hypertrophy, thirteen; generalized hypertrophy associated with prostatic abscess, one; post-prostatectomy scarring of the prostatic urethra, one case. Millin also sets out the following "features" of the operation: (i) It is an extravesical procedure, thus avoiding suprapubic bladder drainage with its risk of slow-closing or persistent fistulæ. (ii) It is applicable to all types of prostatic obstruction. (iii) It is relatively short and shock-free. (iv) It appears to be anatomically sound, no important organs being interfered with or endangered. (v) The mortality is singularly low. No deaths have occurred in the first twenty cases. (vi) The post-operative course is easy for patient and attendant staff. (vii) The whole of the obstructing tissue is removed; so the risk of recurrent obstruction is obviated. (viii) Post-operative stay in hospital seldom exceeds two weeks.

There are some interesting points about Millin's article. The most important obviously is the retropubic extravesical approach. It seems probable from the article that no direct communication is established with the bladder. It would be possible in many instances through an incision in the capsule to enucleate adenomatous tumours growing in the prostate without destruction of the urethra and without damage to the trigonal mucosa. The ejaculatory ducts opening into the prostatic section of the urethra would be torn across, and if the prostatic portion of the urethra was otherwise undamaged after the operation the openings of the ejaculatory ducts would be a potential danger. Millin does not state categorically that the urethra remains intact (except for the damage to the ejaculatory ducts close to the urethra). He does, in discussing reactionary hæmorrhage, refer to a cavity resulting from operation, "closed below by the external sphincter, above by the undamaged internal sphincter, and anteriorly by a firmly sutured incision". The fact that at the end of the operation the catheter has to be guided into the bladder from the prostatic cavity would suggest that the urethra was damaged. Millin also states that in three cases suprapubic leaking of urine occurred owing to faulty catheter fixation. As a matter of fact Millin's subject matter is so important that detail on some of the finer points—a fuller communication—would be welcomed. Another question that must be raised is that of hæmorrhage. Millin seems to have had no trouble with reactionary hæmorrhage, but in two instances secondary hæmorrhage was experienced. No mention is made of sepsis in the cave of Retzius. The risk of such an occurrence will arise at once in the minds of all urological surgeons. The use of sulphanilamide powder, as described by Millin, is, of course, a safeguard, but not an absolute safeguard.

Two points should be noted in regard to Millin's results. First of all he states that it is important that the twenty cases reported by him are a continuous series, with one exception. In this instance the patient suffered from an advanced benign obstruction and had a blood urea content of over 200 milligrammes per hundred millilitres in spite of long-continued catheter drainage; a stab suprapubic cystostomy operation was performed. Millin thus does not adhere solely to his new operation. Again the report states that there were no deaths among a series of twenty patients who were operated on. This is all very well, but there was one case, not included in the series, in which operation was performed "rather to test the safety of the procedure". Presumably in this instance the patient was operated on before any of the twenty with whom the report is concerned. He was a man, aged eighty-four years, who suffered from severe auricular fibrillation, advanced myocardial degeneration, poor renal function and complete urinary retention. Treatment with an indwelling catheter for several weeks had not improved his condition. A large prostate was enucleated successfully by Millin's method, but the patient died from his cardiac condition three and a half weeks later. In regard to this man many will hold, and with considerable justification, that he should not have been excluded from the series; others will hold that in view of his advanced pathological condition, he should not have been operated on at all. As this case was a test case, and Millin proceeded with his series, he cannot have regarded operation as having had anything to do with the patient's death.

Let us now turn to the commendations of *The Lancet*. We read first of all that in this year of 1945, "eighty years after the birth of surgical craftmanship, and in the surgery of the prostate, a branch of operative surgery commoner, more important, more closely studied, and more widely pursued than most, T. J. Millin has discovered a method that is not only quite new, but also simpler, safer, and better than those now in use". Again it is stated that Millin's new operation "seems to avoid the dangers and discomforts of the transvesical, the disasters of the perineal, and the sequelæ of the perurethral operations, and bids fair to supplant them all". One or two of the statements that precede these conclusions should be questioned. The suprapubic operation as generally practised today does, we agree, carry with it certain risks, and it certainly has a considerable mortality in the hands of the "occasional urologist". But such persons as occasional urologists should not be allowed to exist; their surgical consciences should deter them. Such opportunists will be just as dangerous with Millin's operation as they are with any other type of prostatic operation. We read that the Harris operation is an improvement in theory, but that the number of modifications that have been made, and the poor opinion in which they are held by those who have the care of patients afterwards, are evidence of its imperfections. The truth about the Harris operation is that its success is dependent on the meticulous care with which the surgeon follows every detail as described by Harris himself, not the details of any modification. The Harris procedure is the only suprapubic prostatectomy so far devised in which an attempt is made to reform the prostatic urethra—the part of his operation which Harris called "retrigonization". The peculiar arrangement of the sutures in this operation also allows the surgeon to be confident that hæmostasis has been secured. We read further that perurethral operations sometimes cause severe hæmorrhage and that the clinical picture is marred by chances of recurrence and penile stricture. In due time we shall discover whether Millin's operation, which appears to be a good operation, is really as satisfactory as its protagonists would have us believe and whether it has any sequelæ that will mar the clinical picture. Millin himself would have been more convincing if he had been more critical of his own work and had not appeared to gloss over difficulties and imperfections. In all probability we shall find that eventually Millin's operation will become, not the sole method of so-called prostatectomy, but one of several others which will include the Harris operation and perurethral resection and which will be used according to the indications of the particular case under consideration. In the meantime the world of urology can be full of hope.

Abstracts from Medical Literature.

GYNÆCOLOGY.

Uterine Polypi.

K. NEWGARD AND D. G. MORTON (*Western Journal of Surgery, Obstetrics and Gynecology*, August, 1945) discuss polypi of the cervix and of the body of the uterus. They base their study on 228 cases of cervical polypus and 82 cases of endometrial polypus, which occurred at the University of California Hospital between January 1, 1930, and December 31, 1939. Of the patients with cervical polypi, 60·9% were aged between forty and sixty years. Only 99 of the 228 patients had no other pathological condition. Seventy-two patients had no gynæcological complaints at all. The most frequent symptom was hæmorrhage, but the authors point out that 61 of the 76 patients who had profuse hæmorrhage had also some associated pathological condition which would explain the hæmorrhage more readily than the polypus. They believe that it is rare for cervical polypi to cause profuse hæmorrhage. Moreover, additional patients in this group had reached the premenopausal stage, when abnormal hæmorrhage occurs frequently. Other symptoms were vaginal discharge, pruritus, dyspareunia and dysmenorrhœa. Of the 99 patients without some other gynæcological abnormality, 80 had no symptoms directly referable to the cervical polypus. The authors believe that a simple cervical polypus alone rarely gives rise to symptoms, and therefore that if a patient complains of frank hæmorrhage and examination reveals a polypus, care should be taken to exclude other causes for the hæmorrhage. Of the 82 patients with endometrial polypus, 34 were aged between forty-one and fifty years. In 31 instances the polypus was purely an accidental finding, no symptoms being present. The most common symptom was hæmorrhage of an intermittent type. In eight cases a watery, sometimes purulent, discharge was present as well, and in five cases a discharge was present without the hæmorrhage. In the remaining cases dysmenorrhœa or menorrhagia was present, which may or may not have been due to the polypus. Twenty-seven of the patients were in the menopausal stage and had ceased to menstruate. In 58 cases a variety of concomitant pathological conditions was present. Both types of polypus form tumour-like growths which have to be strictly separated from other polypoid masses in the cervix or uterine cavity. Their structures respectively resemble those of the cervical and uterine mucosa. Secondary changes are frequent and involve epithelium and stroma. The most important changes are non-malignant epidermization in cervical polypi and malignancy in endometrial polypi. The authors believe that the too loose employment of the term polypus explains many of the so-called malignant cervical polypi reported in the past. While polypoid carcinoma of the cervix occurs, and while rarely a true polypus may become malignant, these changes are usually evident on clinical examination. A simple cervical mucous polypus may be removed in the surgery, if it is certain that no serious associated abnormality is present and if the patient's symptoms can be explained by the presence of the polypus. The treatment of choice is by excision and cauterization of the base.

Coexisting Tuberculosis and Cancer of the Uterus.

G. HARVEY SMITH (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1945) records a case of tuberculosis and cancer coexisting in the uterus. The patient was aged forty-four years. While she was undergoing an operation for appendicectomy, it was found that the uterus was enlarged and that both ovaries were small and atrophic. No evidence of tuberculosis was found. Progress was uneventful, and the patient was referred for gynæcological examination, which revealed that the uterus was anterior, soft, globular and about the size of a grapefruit, and that the ovaries were not palpable. The patient had for seven months had a yellow vaginal discharge, which was relieved by pessaries given by her doctor. Four months after the cessation of the discharge she was admitted to hospital for investigation; she had had a sudden gush of thick yellow discharge from the vagina, followed by blood-stained fluid. This was preceded and followed by severe abdominal pain. Extreme tenderness was present above the *symphysis pubis*, and a mass was palpable through the anterior fornix. After examination under anæsthesia no further treatment was considered necessary. Three months later the patient was readmitted to hospital with severe abdominal pain, frequency of micturition, and hæmorrhage *per vaginam* of three weeks' duration, the blood now coming in clots. Subtotal hysterectomy was performed and the right adnexa were removed. White nodules were present at the uterine end of both Fallopian tubes, and the ovaries appeared healthy. A growth was found in the posterior uterine wall, protruding into the cavity. The diagnosis of carcinoma of the body of the uterus with metastatic deposits in both Fallopian tubes was made. Microscopic examination of transverse sections of the part of the Fallopian tube first thought to be affected by metastasis, revealed that the nodule was the site of caseating tuberculous salpingitis with well-formed giant-cell systems. No tumour cells were seen, and no evidence of peritoneal involvement was obtained. Histological study of the uterine tumour led to the conclusion that it was an anaplastic adenocarcinoma of the body of the uterus. Tubercle follicles were found in the tumour mass itself and in continuity with the neoplastic process. The tubercles were largely non-caseous and evidently fairly young. Their distribution and development suggested that they were younger than the tumour, and were probably the result of dissemination from the older tubal lesion. There was no evidence to suggest that the tuberculous process had any bearing on the origin of the tumour. The association of the two lesions was thought to be accidental. Attempts to discover tubercle bacilli in the lesions by the Ziehl-Neelsen method were unsuccessful. Neither pulmonary nor renal tuberculous lesions could be found in the patient, and she left hospital in good condition. When the report was written, no further trouble had been experienced.

Extrophy of the Bladder with Procidentia.

S. G. CLAYTON (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1945) reports a case of extrophy of the bladder with procidentia. He states that the condition is rare and presents a difficult problem. The patient was aged thirty-one years and had already "endured much surgery"; the lower part of the abdominal wall was represented only by an attenuated scar. The ureters had been transplanted into the bowel. The pubic rami and labia were widely separated, and the position of the symphysis and *mons Veneris* was occupied by a narrow bridge of skin and fibrous tissue. The perineum was normal. At times the cervix protruded outside the vaginal orifice, and the anterior vaginal wall was everted below this bridge of skin. A cup and stem pessary was used to control the prolapse. Three years previously right renal calculi had been removed by pyelolithotomy; but nephrectomy became necessary seven months prior to the patient's coming under observation. The author states that when he first examined the patient she was complaining of severe discomfort due to the prolapse. Her general condition was good. Her urine could not be examined, as it was discharged rectally, but there was no evidence of renal infection, and excretion pyelography revealed good excretion from a normal left kidney. She could hold her urine in the rectum for two hours. The prolapse was complete; the whole vagina was everted, and severe infection and crusting over the mucosa were present. She could not use her cup and stem pessary or walk or sit in comfort. Her menstrual periods were regular and normal. She welcomed an attempt at surgical cure. The possible operative procedures were limited by the patient's previous surgical adventures. At operation under nitrous oxide, oxygen and ether anæsthesia, the posterior fornix was incised and the pouch of Douglas was found to be obliterated by adhesions. The posterior wall of the uterus was followed by gentle dissection, the distended rectum being pushed backwards and freed from the fundus of the uterus, where denser adhesions were encountered. The Fothergill incision was made down the anterior vaginal wall and around the cervix, and the anterior wall of the uterus was dissected free from the fibrous tissue that occupied the normal place of the bladder. The broad ligaments were defined and clamped and the uterus was removed. The broad ligaments were sewn together, and the usual repair of the anterior wall was performed. In view of the gross local sepsis and of fear for the vitality of the rectal wall from the uterine adhesions, some packing was placed in the posterior fornix, which was not completely sutured but left to granulate. A rectal tube was passed to drain urine from the bowel. Recovery was uneventful, and the patient afterwards married. Some bulging of the anterior wall remained, and this later appeared to be increasing. A modified ball and stem pessary was

able to be worn with comfort. The author considers that if the eversion progresses, some type of le Forte procedure will have to be performed, and that the ultimate prognosis depends on the fate of the remaining kidney. A few attacks of pain in the loin and fever suggest some ascending infection.

Extragenital Chorionepithelioma.

L. RESNICK (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1945) discusses extragenital chorionepithelioma, and reports a case in which chorionepithelioma of the breast occurred during pregnancy. He also presents a review of the literature. The author states that chorionepithelioma has been classified into two main groups: (i) a genital group, which is further subdivided into (*a*) uterine and (*b*) extrauterine varieties, and (ii) an extragenital group, defined as chorionepithelioma manifesting itself in organs of the body remote from, and without microscopic evidence of a similar growth in, the genital tract. In respect of the clinical relationship of the condition to pregnancy, three types may be distinguished: (i) that in which the disease develops during pregnancy—the commonest site appears to be the vagina; (ii) that in which the disease develops after an interval of several months to several years—the majority of cases of both the genital and extragenital varieties fall into this group; (iii) that in which the clinical relationship to pregnancy is obscure—in this group can be included the cases of so-called teratomatous origin. In the sixteen cases so far recorded, the distribution has been as follows: thirteen tumours in the lungs, ten in the liver, eight in the kidneys, seven in the intestines, five in the brain, two in the suprarenals, spleen and thyreoid gland, and one each in the mediastinum, heart, pancreas and breast. Four theories of causation are stated: (i) transportation of the chorionepitheliomatous emboli from a previous chorionepitheliomatous placenta completely expelled during labour; (ii) malignant transformation of transported chorionic emboli from a normal placenta; (iii) complete regression of the uterine tumour with continued growth of the metastases in distant organs; (iv) development from the trophoblast of an extragenital teratoma.

OBSTETRICS.

"Prostigmin" as a Test for Early Pregnancy.

A. A. CORBER (*Canadian Medical Association Journal*, July, 1945) advocates the use of "Prostigman" in cases of delayed menstruation. He maintains that this will induce menstrual bleeding in all but a few patients unless pregnancy exists. If the patient is pregnant, no bleeding will occur and no damage will be done. The author summarizes the rationale for this treatment by explaining that "Prostigmin" inhibits cholinesterase, thus enhancing the effect of acetylcholine in producing uterine hyperæmia. When the primary stimulating agents, that is, the œstrogens, are present the injection of "Prostigmin" sets into action the secondary vascular effects. Cases in which the treatment will fail are those in which there is an underlying endocrine disturbance, or else some general debilitating disease or those in which local pelvic disease is present. "Prostigmin" is therefore not only valuable in the treatment of delayed menstruation, but is a useful test for early pregnancy.

The Influence of Stilbœstrol on Lactation.

HENRY FIELDS (*American Journal of Obstetrics and Gynecology*, March, 1945) was stimulated by the differences of opinion concerning the efficacy of stilbœstrol in suppressing lactation to make a study of 250 patients. He particularly wished to compare its effects on mothers with no nipple stimulation and on those whose breasts were emptied either by suckling or pumping during the progress of treatment. In all cases studied he used none of the usual supplementary measures for inhibiting lactation. The mothers were given a normal fluid intake and no undue cathartics, and the breasts were properly supported with no binding. The dosage of stilbœstrol was one milligramme three times daily for five days. Under these conditions, he found a definite suppression of lactation in mothers with no nipple stimulation, in those with associated pumping of the breasts and in those who had previously fully established lactation by a short period of nursing. Furthermore a group treated while nursing their babies showed a definite decrease in milk supply during the administration of stilbœstrol. In this group the milk supply returned to normal after treatment was discontinued. Another point is the ability of women to take *post partum* varying doses of stilbœstrol by mouth with no untoward effects. None of the mothers or infants in the series showed any unusual signs or symptoms. Furthermore it was found that one milligramme of stilbœstrol taken three times daily produced the same degree of milk suppression as twenty milligrammes taken four times daily.

Anæmia in Pregnancy.

JOHN WOLFF AND LOUIS R. LIMARZI (*The Journal of the American Medical Association*, June, 1945) discuss the blood picture in normal pregnant women and compare it with the findings in anæmic pregnant women. Their studies include both peripheral blood and bone marrow. They agree with other observers in noting a constant decrease in erythrocytes and hæmoglobin value from the third or fourth month of pregnancy. They attribute this to a relative increase of plasma volume rather than to a lack of iron, as the bone marrow picture shows no change in the character of normal erythropoiesis. Some workers are quoted as stating that hæmoglobin, ten grammes, and erythrocytes, 3·36 million per cubic millimetre, are the minimal normal figures during pregnancy. Others give higher values up to 11·3 grammes and 4·0 million respectively. Examples are studied of hypochromic, microcytic and normocytic anæmias which responded well to iron therapy. The bone marrow in these cases showed a normoblastic hyperplasia. The key to iron therapy is the mean corpuscular hæmoglobin concentration. When this is below normal it proves the existence of a true hypochromic anæmia rather than a so-called physiological anæmia. The third type of pregnancy anæmia is the megaloblastic or pernicious anæmia of pregnancy. This differs from the true pernicious anæmia in the lack of associated neurological symptoms, smooth tongue and splenomegaly. Also no achlorhydria is found. The blood picture here is that of a macrocytic, hyperchromic or hypochromic anæmia, and it is usually of a more severe character than the other types. The bone marrow shows a megaloblastic type of erythropoiesis and some abnormal granulopoiesis. This anæmia seems to be found always in the latter months of pregnancy. It requires large and frequent intramuscular doses of potent liver extract and several blood transfusions to bring about a satisfactory recovery phase. The peripheral blood and bone marrow recover completely and permanently in about five months after delivery. The infants have normal blood and bone marrow, as also do other children of the mother. In conclusion, the authors state that the most accurate criterion for the diagnosis and treatment of the true anæmia of pregnancy is the study of the bone marrow. The changes in the peripheral blood are of a secondary nature and may be misleading.

Uterine Defence Mechanism against Infection.

J. R. GOODALL (*American Journal of Obstetrics and Gynecology*, April, 1945) has studied the mechanism by which infection is avoided in the uterus after abortion or confinement. He considers that the uterus has a special defence mechanism, some aspects of which are known—desquamation, exfoliation, local lymphocytosis, development of mesenchymal cells in the parametrial tissues during pregnancy (Hofbauer's cells), and closure of the uterine cavity by blood clot. There are probably others of a biochemical nature not yet known or understood. The author discusses the role of each of these factors, and concludes that they together form a strong barrier against invasion by pathogenic organisms. The sealing of the cavity by blood clot and the growth of a new mucosa under the bridgework seal the open wound. When the cavity is completely closed off, the major part of the lochia comes from the lower uterine segment and from the serum squeezed out of the uterine cavity during the early days *post partum* and from the disintegration of the uterine fibrin after it has fully discharged its function.

The Transmission of Penicillin through the Placenta.

A. M. HUTTER AND J. PARKES (*American Journal of Obstetrics and Gynecology*, May, 1945) have investigated the blood level of penicillin in the mother and the fœtus when the drug is given to the former and report their findings. It was found that 100,000 units of penicillin injected intramuscularly into the pregnant patient at term would produce an adequate bacteriostatic penicillin level in the fœtal circulation. This opens up a rapid method of treatment of syphilis and gonococcal infections; penicillin given during labour should reduce fœtal infection in cases of prolonged labour with ruptured membranes.

Special Article.

RESEARCHES ON "PALUDRINE" (M.4888) IN AUSTRALIA.

From the Land Headquarters Medical Research Unit, Cairns.[1]

ANY army possessing an antimalarial drug which would completely prevent malaria from developing in troops operating in highly malarious areas would have a tremendous advantage over the enemy. For this reason, the results of researches on antimalarial drugs in general had to be secret, especially following the entry of Japan into the war. For security reasons newly discovered chemical compounds were referred to only by code numbers, and the great majority of research workers knew little or nothing about the chemical formulæ of the antimalarial drugs they were investigating.

HISTORY OF THE INVESTIGATIONS.

From 1942 onwards in England research chemists and biologists in the laboratories of Imperial Chemical Industries succeeded in synthesizing a unique series of new antimalarial compounds of increasing potency. The earlier drugs in the series were interesting, but not good enough to justify extensive trial on human beings. In 1944, however, Mr. F. M. Rose, working in collaboration with Mr. F. H. S. Curd, synthesized two new drugs which Dr. D. G. Davey, who was responsible for all the biological testing on bird malaria in this research, regarded as most promising. He found not only that these two drugs destroyed malaria parasites in the blood of birds, but that in the species of bird malaria tested (*Plasmodium gallinaceum*) they actually prevented the infection from developing. In other words, these drugs were acting as true causal prophylactics. Early in 1945, Dr. Davey and his team had found one of these drugs (M.4888) definitely superior to the other (M.4318), inasmuch as M.4888 prevented malaria from developing in several different species of malaria affecting birds—*Plasmodium gallinaceum, Plasmodium relictum, Plasmodium cathemerium* and probably *Plasmodium lophuræ* as well. This lead to the hope that it might prove to be a causal prophylactic in human malaria also.

Unfortunately, as experience in the United States of America, Great Britain and Australia has shown during the present war, it does not necessarily follow that a drug which will prevent avian species of malaria parasites from developing in birds, will prevent human species of malaria parasites from producing malaria in man.

This is well illustrated by reference to sulphamerazine, which was found in 1942 by biologists in the United States of America to be a true causal prophylactic in *Plasmodium gallinaceum* infection in chicks. Field reports on the prophylactic value of similar sulphonamides in troops in West Africa, where *Plasmodium falciparum* infection predominates, appeared to support a similar action in human malaria. Subsequent critical investigations on experimentally infected volunteers at Cairns in 1943 proved that: (i) Sulphamerazine and the allied sulphonamides, even in malignant tertian malaria, were not true causal prophylactics in man, but acted as schizonticides. (ii) While they suppressed and cured some 85% of malignant tertian infections when given in a dosage of 1·0 gramme daily, they completely failed to cure and in most instances completely failed to suppress *Plasmodium vivax* infections. As both species of malaria are prevalent here, sulphamerazine was obviously unsuited for the South-West Pacific Area.

The Medical Research Council, which controlled all malaria research in Great Britain, arranged early in 1945 for clinical trials on malaria patients and studies on the pharmacology of "Paludrine" to be carried out at the Liverpool School of Tropical Medicine in England and for experiments on human volunteers to be undertaken by the Land Headquarters Medical Research Unit at Cairns, especially from the viewpoint of causal prophylaxis and action on parasitic strains found in the South-West Pacific.

[1] Director, Brigadier N. Hamilton Fairley; Commanding Officer and Senior Physician, Lieutenant-Colonel C. R. Bickerton Blackburn; Physician, Captain R. H. Black; Pathologists, Major T. S. Gregory, Major J. I. Tonge; Assistant Pathologists, Lieutenant K. S. Pope, Lieutenant S. R. Dunn, Lieutenant M. S. A. Swan and Lieutenant T. A. F. Akhurst; Entomologist, Major M. J. Mackerras; Assistant Entomologists, Lieutenant T. H. Lemerle and Lieutenant Q. N. Ercole.

The British work on "Paludrine" was reviewed in the *British Medical Journal* on November 10, 1945, at page 653, but owing to a sudden decision to release information on M.4888 and rename the drug "Paludrine", it was not possible in the time available to include an up-to-date account of the Australian researches. In 1944 the name "Paludrine" had been given by Imperial Chemical Industries to an early and less effective antimalarial compound of the same series (M.3349). This led to a certain amount of confusion, for when the British Press and radio announced the discovery of "Paludrine" and stated that this drug was being extensively investigated in Australia an official denial was made from headquarters of the Australian Military Forces. At that time no notification had reached headquarters of the Australian Military Forces that the security silence had been lifted on M.4888 or that it had been renamed "Paludrine".

RESEARCHES AT LAND HEADQUARTERS MEDICAL RESEARCH UNIT, CAIRNS.

Approximately 200 volunteers have been exposed to experimental malaria infection in connexion with this research, and the drug has been or is being used for clinical trials on several hundred patients suffering from malaria contracted in New Guinea or elsewhere in the South-West Pacific Area.

Two species of malaria commonly affect troops in the tropics, the dangerous malignant tertian malaria caused by *Plasmodium falciparum*, which rapidly kills if not treated, and the far less dangerous but more troublesome relapsing type of benign tertian malaria caused by *Plasmodium vivax*. A drug which prevents or cures one of these species may fail to do so with the other; in consequence each species has to be investigated separately.

Four problems concerning "Paludrine" have been and are being critically investigated at Cairns, and until these have been answered the full value of this drug cannot be finally assessed. These problems are: (i) Is "Paludrine" a true causal prophylactic? (ii) Is it an efficient schizonticide and does it produce clinical cure? (iii) Will it result in radical cure? (iv) Is it gametocidal or will it prevent anopheline mosquitoes, feeding on gametocyte carriers taking the drug, from becoming infective?

Is "Paludrine" a Causal Prophylactic?

Is "Paludrine" a causal prophylactic? In other words, does this drug prevent the parasites of malignant and benign tertian malaria from developing in man by acting on the sporozoites or exo-erythrocytic or e.e. forms.

Malignant Tertian Malaria.

The first important result obtained at Cairns has been to show that in malignant tertian malaria "Paludrine" destroys the parasites inoculated by the infected mosquito at an early stage of their development before they regain access to the circulating blood.

So potent is this drug that if 1·0 gramme of "Paludrine" is swallowed three hours before the volunteer is bitten by ten heavily infected anopheline mosquitoes, malignant tertian malaria does not occur. Similarly a single dose of 0·1 gramme, given on the second, third, fourth or fifth day following infection, prevents malignant tertian malaria developing. This simple type of experiment definitely proves that "Paludrine" prevents malaria and is a true causal prophylactic in *Plasmodium falciparum* infections. No matter how much quinine or "Atebrin" is given in identical circumstances, malignant tertian malaria invariably follows. The only drug with a comparable action is "Plasmoquine", and to get this result it has to be used in toxic dosage.

In the field the customary military procedure to suppress malaria is to give one tablet of a drug every day. It has been found at Cairns that no matter how many mosquitoes (*Anopheles punctulatus punctulatus*) infected with *Plasmodium falciparum* bit volunteers, the volunteers failed to develop the slightest evidence of malaria, provided they were taking one tablet (0·1 gramme or 1·5 grains) of "Paludrine" every day. During their field tests these volunteers were subjected to repeated malarial infections, and to long marches in hilly country in a tropical climate—thirty miles in one day, eighty-nine miles in three days, and finally seventy-two miles in thirty-six hours. They also voluntarily subjected themselves to extreme cold in the freezing chamber of the local meat works, staying in for one hour at −10° C. Despite these and other stresses, they remained perfectly fit. Fever was entirely absent, the spleen never enlarged, and malaria

parasites could not be demonstrated in the blood even after the most searching microscopic examinations lasting one hour or longer.

As it is feasible to examine only a very small quantity of blood (1·0 millilitre) under the microscope, the possibility remains that parasites may be there in numbers too scanty to be seen, that is, in a submicroscopic density. To exclude this possible fallacy 200 millilitres of blood (that is, 200,000 times as much as it is practicable to examine microscopically) were taken from these volunteers and injected into other volunteers to ascertain if the recipients got malaria. None did. With the Julian Smith rotary blood transfusion pump, it was possible to prove that malaria parasites were not present in the circulating blood of the original volunteers, even though they had been bitten by many infected mosquitoes known to be carrying sporozoites of *Plasmodium falciparum* in their salivary glands.

"Atebrin"—the drug which has enabled us to maintain our armed forces fighting against the Japanese in highly malarious areas in the Pacific and East Asia Commands—does not act in this way. If an individual taking "Atebrin" every day is bitten by infected mosquitoes, parasites appear in submicroscopic numbers in the blood from the seventh day onward, but if "Atebrin" is present in sufficient concentration, the parasites are destroyed within the next three to five days and cure of the malignant tertian malaria infection results. When taken in a dosage of one tablet (0·1 gramme) every day, the difference between "Paludrine" and "Atebrin" is that with "Atebrin" the asexual parasites are destroyed after reaching the circulating blood, whereas the exo-erythrocytic parasites (e.e. forms) are destroyed by "Paludrine" before they can produce erythrocytic parasites.

Benign Tertian Malaria.

If volunteers taking one tablet of "Paludrine" (0·1 gramme) daily are bitten by mosquitoes infected with *Plasmodium vivax*, they fail to develop symptoms of malaria or show malaria parasites in the blood while "Paludrine" is being taken. Some time after they cease to take the drug, however, parasites may appear and a frank malarial attack may develop.

Somewhat similar findings occur in volunteers exposed to infection while taking one tablet of "Atebrin" every day, except that though generally parasites cannot be found in the blood with the microscope, they can be demonstrated invariably by injecting into another volunteer 200 millilitres of blood collected nine days after exposure to infection. If the infected volunteer is taking "Atebrin", the recipient of his blood goes down with an attack of benign tertian malaria, but if he is taking "Paludrine", malaria does not develop.

Though "Paludrine" inhibits the development of benign tertian malaria parasites in the host, prevents their appearance in the blood and suppresses fever while the drug is being taken, it acts only as a partial causal prophylactic in *Plasmodium vivax* infections and an attack is liable to develop once the drug is stopped.

The reason for this evidently lies in the persistence of exo-erythrocytic parasites (e.e. forms) which have survived, despite the fact that their cyclic development had been inhibited throughout the period of "Paludrine" administration.

Is "Paludrine" a Satisfactory Schizonticide?

"Paludrine" has proved a satisfactory schizonticide for the control of the clinical attack of malaria in both malignant tertian and benign tertian infections. Researches at Cairns indicate that it inhibits nuclear division in the early schizont stage, causes degeneration of asexual parasites before developing into mature schizonts and gives clinical results which are comparable but in no way superior to those obtained with appropriate doses of quinine or "Atebrin". In *Plasmodium falciparum* infections its action in inhibiting nuclear division has been demonstrated by Captain R. H. Black by cultivating the parasites *in vitro* in a blood glucose serum medium containing the drug.

One remarkable feature of "Paludrine" as a schizonticide is the smallness of the dose required to control an attack of malaria fever. One tablet (0·1 gramme) has proved sufficient to end the attack and bring the temperature to normal in both malignant tertian and benign tertian infections. Nothing approaching this can be achieved by either quinine or "Atebrin", while "Plasmoquine" is quite inferior as a schizonticide and even in toxic dosage fails in *Plasmodium falciparum* infections.

Does "Paludrine" Produce Radical Cure?

Does "Paludrine" produce radical cure? In other words, does "Paludrine" produce a permanent cure so that an infected individual who returns to a non-malarious area and undergoes a course of treatment will have no further attacks?

At Cairns, out of 65 patients suffering from malignant tertian malaria treated, 64 have been proved to be cured. The course of treatment used was short, consisting of three tablets daily for ten days. This is a remarkable result and superior to what could be achieved with either quinine or "Atebrin" in the same period.

It is too early yet to assess the ultimate cure rate in benign tertian malaria, as sufficient time has not elapsed to be sure that delayed relapses will not follow.

In one group of 31 volunteers with primary benign tertian malaria, however, only five out of thirty-one have suffered a relapse to date. Ten tablets (1·0 gramme) were given every day for fourteen days to these volunteers, and despite the large dosage little inconvenience resulted. Even, however, if "Paludrine" fails to cure most individuals with relapsing benign tertian malaria, the results at Cairns suggest that the mere taking of one or two tablets each week would give complete freedom from attacks.

Recently for the first time a relatively "Atebrin"-resistant strain of *Plasmodium falciparum* has been studied at Cairns which requires 0·2 gramme of "Atebrin" daily to suppress symptoms in experimentally infected volunteers and even then may fail to effect a cure. This strain shows no enhanced resistance to quinine, sulphamerazine, "Resochin" (S.N.7618) or "Paludrine". Indeed its sensitiveness to "Paludrine" was remarkable, for it was found in repeatedly infected volunteers that one-quarter of a tablet (0·025 gramme) daily was a complete causal prophylactic. In other words one-eighth of the dose of "Paludrine" was completely effective, yet in equivalent dosage "Paludrine" is much less toxic than "Atebrin".

Another experiment on 600 cases of relapsing benign tertian malaria is in progress at 113 (Concord) Military Hospital, 115 (Heidelberg) Military Hospital and 112 (Brisbane) Military Hospital, and is designed to ascertain whether the combination of "Paludrine" (0·3 gramme daily) with "Plasmoquine" base (0·03 gramme daily) for ten days is superior to quinine (30 grains) and "Plasmoquine" base (0·03 gramme daily) for ten days. There is evidence that both "Paludrine" and "Plasmoquine" act on the persistent (secondary) e.e. forms of *Plasmodium vivax*, and it is possible that the combination of the two drugs may produce a higher proportion of radical cures than any other combination of antimalarial drugs.

The Effect of "Paludrine" on Gametocytes.

Plasmodium Falciparum.

The question of the effect of "Paludrine" on the gametocytes of *Plasmodium falciparum* has been investigated by Major M. J. Mackerras and Lieutenant Q. N. Ercole at Cairns.

Gametocyte production is apparently not checked by "Paludrine", since after a full course (0·3 gramme daily for ten or fourteen days) patients exhibit gametocyte waves, the height and duration of which fall within normal limits.

If the drug (either in doses of 0·1 or 0·3 gramme daily) was given when gametocytes were already present, they persisted in the circulation morphologically unchanged for a normal period. However, if mosquitoes were fed on a patient taking the drug no salivary gland infection was ever recorded.

Complete sterilization of the infection occurred in mosquitoes fed on a gametocyte carrier one hour after the first dose of 0·1 gramme of "Paludrine" was taken.

The drug did not produce any irreversible change in the gametocyte, but killed the protozoon only after it had begun development in the mosquito. This was shown by the fact that, when gametocytes were sufficiently numerous and persisted after the cessation of therapy, they might recover completely when the drug was eliminated.

After the initial rise gametocytes tend to decrease gradually in numbers and infectivity, irrespective of therapy (except, of course, "Plasmoquine" therapy), and one may expect infections to get lighter if anything from day to day. After cessation of "Paludrine", however, infections of increasing heaviness are obtained.

When mosquitoes were fed on carriers two, four, five and six days after the cessation of therapy (0·3 gramme daily for ten days), no infection was recorded. On the seventh,

eighth and tenth days oocysts formed, but failed to grow. On the twelfth, thirteenth and fourteenth days oocysts developed normally and sporozoites invaded the salivary glands. The end of the infection in mosquitoes fed on the tenth day resembled that seen on the first day of therapy, that is, death and shrivelling of the young oocyst. Apparently traces of the drug sufficient to kill the organism were still present on the tenth day, but had disappeared on the twelfth day after the cessation of therapy.

There is evidence that the effects of a single dose of 0·1 gramme cease by the fourth day. Gametocytes taken up by mosquitoes four or more days after the administration of 0·1 gramme proved capable of completing their development.

Plasmodium Vivax.

It appears that "Paludrine" has no effect on the numbers or morphology of *Plasmodium vivax* gametocytes. In wet preparations made from one to five days after the beginning of therapy many gametocytes appeared quite normal with pigment in characteristically rapid motion. Exflagellation with the formation of vigorous microgametes was observed in one case on the fifth day of therapy (0·3 gramme daily) and in another on the fourth day of increased dosage (1·0 gramme daily).

When anophelines (*Anopheles punctulatus punctulatus*) were fed on gametocyte carriers who had received a single dose of 0·15 gramme or 0·2 gramme, exflagellation, fertilization and vermicule formation occurred normally. The vermicules penetrated the gut wall and became encysted. With one exception development in every batch of mosquitoes was arrested at this point, and two weeks after feeding when the cysts should have been mature, all that could be found in the gut wall were small shrunken cysts containing a few clumps of pigment. In the exception referred to mosquitoes were fed on the carrier four and a half hours after his first dose. In about 50% of those dissected some cysts had grown a little. The two largest seen measured 3μ by 25μ and 22μ by 20μ (that is, approximately half-grown). No further growth took place and the cysts ultimately became shrunken and some became chitinized.

In mosquitoes fed on the second day of therapy (0·3 gramme daily) development occurred normally up to the vermicule stage, but penetration of the gut wall was not observed.

Exflagellation and fertilization were observed in three cases on the third day and in one case on the fourth day of therapy, but vermicules were not detected and no cysts appeared in the gut wall.

Trophozoites had usually disappeared from the blood by the third or fourth day and gametocytes by the seventh or eighth day of therapy.

SUMMARY.

Though "Paludrine" has no demonstrable effect on the gametocyte and is not a gametocidal drug in the accepted meaning of the term, complete sterilization of the infection occurs in mosquitoes fed on a gametocyte carrier as early as one hour after 0·1 gramme of the drug is taken. This sterilization effect persists for a variable number of days after medication ceases and depends on the dosage administered. From a practical point of view it can be stated that a ten-day course of "Paludrine" (0·3 gramme daily) is quite effective in controlling the infectivity of the gametocytes in benign tertian malaria, and that if this treatment is prolonged for three weeks in malignant tertian malaria a similar result will be achieved.

The standard clinical course likely to be adopted in the Australian army is one tablet (0·1 gramme) thrice daily after food for three weeks, or one tablet of "Paludrine" (0·1 gramme) and one tablet of "Plasmoquine (0·01 gramme) thrice daily for ten days.

If *Plasmodium vivax* relapses still occur, the individual will be advised to take one tablet of "Paludrine" (0·1 gramme) twice weekly indefinitely or until cure has probably been attained.

For suppressive purposes in the field one tablet (0·1 gramme) twice a week, that is, at intervals of three or four days, should give complete protection. The Cairns experiments indicate that in this dosage the drug acts as a true causal prophylactic in malignant tertian malaria and completely suppresses benign tertian malaria. Whether, in this dosage over long periods of time, it will cure benign tertian malaria by its action in preventing nuclear division in e.e. forms remains to be determined.

CONCLUSIONS FROM THE CAIRNS EXPERIMENTS ON "PALUDRINE".

The results on experimentally infected volunteers at Cairns and clinical trials with "Paludrine" in various Australian military hospitals have confirmed and extended in human malaria the results obtained by Dr. D. G. Davey and his team in bird malaria.

1. "Paludrine" is superior to all known antimalarial drugs inasmuch as in non-toxic dosage it is a true causal prophylactic in malignant tertian malaria and a partial causal prophylactic in benign tertian malaria. The only other drug which has a similar action is "Plasmoquine"; but this drug has to be given in a dosage which is too dangerous for routine use in man.

2. "Paludrine" in therapeutic dosage controls malaria fever and terminates the clinical attack. It produces radical cure in malignant tertian malaria. The percentage of radical cures in benign tertian malaria is not yet known, but the Cairns results suggest that when individuals are not cured, the administration of 0·1 gramme twice weekly indefinitely would prevent relapses until cure was attained.

3. Though "Paludrine" is not gametocidal in the accepted meaning of the term, sterilization of the infection occurs in mosquitoes fed on a gametocyte carrier as early as one hour after the drug is taken. This sterilization effect persists while the drug is being taken and for a variable period after administration of the drug ceases, depending on the dosage adopted. Its action in this respect is superior to that of quinine or "Atebrin", for gametocyte carriers may continue to infect mosquitoes while these drugs are being administered. On the other hand, as a true gametocide "Paludrine" is inferior to "Plasmoquine".

4. The difference between the effective therapeutic dose and the toxic dose is very considerable. Taking this into consideration, "Paludrine" is undoubtedly the most potent antimalarial drug known. Its discovery is a triumph for British chemotherapy.

British Medical Association News.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed or will resume practice as from the dates mentioned:

Dr. H. R. Love, Wickham House, Wickham Terrace, Brisbane (February 1, 1946).
Dr. R. G. Quinn, Lutwyche Road, Windsor, Brisbane (January 8, 1946).
Dr. B. R. Morey, 56, Macquarie Street, Parramatta, New South Wales.
Dr. W. C. B. Harvey, 135, Macquarie Street, Sydney (January 2, 1946).
Dr. G. S. Flynn, 135, Macquarie Street, Sydney (February 1, 1946).
Dr. Henry Stephens, 85, Spring Street, Melbourne (December 17, 1945).
Dr. H. M. Fisher, 177, North Terrace, Adelaide (March 1, 1946).
Dr. W. A. Dibden, 172, North Terrace, Adelaide (February 18, 1946).
Dr. Mark Bonnin, 178, North Terrace, Adelaide, South Australia.

Special Correspondence.

CANADA LETTER.

BY OUR SPECIAL CORRESPONDENT.

THE end of the year 1945 has brought a general stock-taking in Canadian medicine. It may be fair to say that no single year in our history has witnessed such an orientation of public discussion in the subjects of medical care, prepayment plans of health insurance, and medical education. The remarkable work of the National Research Council has appealed strongly to the imagination of a people now very

research conscious due to Canada's connexion with the atomic bomb.

One of the year's major accomplishments has been the setting up and staffing of a Ministry of National Health and Welfare under a capable and enthusiastic young minister, the Honourable Brooke Claxton. The former Director-General of Medical Services for the Army, General Brock Chisholm, has been made the Deputy Minister in charge of health, while Dr. George Davidson, formerly Director of Social Welfare for the Province of British Columbia, has been made Deputy Minister in charge of welfare.

Amongst the subjects occupying the new ministry's attention is that of old-age pensions. Since it is estimated that within thirty years one person in fifteen in Canada will be over the age of seventy, there is a keen concern at Ottawa in the drafting of sound fiscal policy so that this burden may be shouldered by the national government without delay.

An innovation in public health and physical fitness has been introduced in Saskatchewan, where the Director of Physical Fitness for the province, Dr. J. B. Kirkpatrick, an air force veteran, has developed a programme of physical fitness in the jails of the province. The psychiatric value of the programme is becoming strikingly manifest.

Health and welfare services together with education in the Province of British Columbia now absorb approximately one-half of the annual budget of that rapidly expanding western area. Though its university is the youngest in Canada, it now has 7,000 students, and with the opening of a medical school in the near future will be Canada's second in size. While the province has had a most vigilant venereal disease administration, the mass movement of population during and since the war has made the control of disease most difficult. In fact, the first six months of 1945 showed an increase of 100% over the same period in 1944 in primary and secondary syphilis. The treatment of such cases has posed a problem for the authorities directing the discharge of personnel from the forces. Fever therapy combined with massive dosage of penicillin offers a solution only in so far as the treatment is continuous. The restlessness engendered in the population with the end of hostilities has added to the difficulty of ensuring completion of treatment. An additional distraction has been added by the unfortunate conception popularized by the *Reader's Digest* of the efficacy of a "one-day treatment for syphilis". Unfortunately the lay public of Canada and the United States is keener on pseudo-science than medical accuracy at the moment, and it will indeed take time to counteract the effect of the *Reader's Digest* in medicine and in other fields.

The Montreal Neurological Society closed a year of excellent meetings with a memorial to the late Professor del Rio-Hortega, who died in June in the Argentine as an exile from Spain. His contributions to neuropathology were reviewed by Dr. Wilder Penfield, Professor Pierre Masson, and del Rio-Hortega's first pupil, Dr. Miguel Prados. A symposium on the care of paraplegic veterans was also held, at which social workers and neurosurgeons contributed. Canada has a large programme for the rehabilitation of these men from the armed forces.

The future methods for the prepayment of medical care are being worked out by an economic commission at Ottawa representing Federal and Provincial Governments.

Correspondence.

TUBERCULOSIS PATIENTS AND HOSPITALS.

SIR: In your issue of January 5, 1946, Dr. D. B. Pitt inquires if any provision is made in Melbourne for an ambulant pneumothorax clinic.

With the concurrence of Dr. J. Bell Ferguson, State Director of Tuberculosis, it has been decided to commence such a clinic at the Central Hospital, Melbourne. Five male and five female beds will be retained for cases requiring artificial pneumothorax. Inductions will be carried out whilst the patients are in hospital, and so soon as the collapse is stabilized, the patient discharged to home, the refills being maintained at the Refill Clinic. Patients admitted to these beds will be short-term in-patients, and if artificial pneumothorax is not feasible, or cannot be satisfactorily maintained, there can be no undertaking to retain the patient.

Admissions will, as at present, be arranged by the Tuberculosis Bureau, but admission may be expedited by, at the same time, notifying the Medical Director, Central Hospital, of suitable cases for ambulant pneumothorax therapy.

It must be appreciated that this arrangement is an emergency measure, designed solely to relieve the acute position in tuberculosis accommodation at present existing in Victoria.

Yours, etc.,

ALAN H. PENINGTON, M.D.

Central Hospital,
172, Lonsdale Street,
Melbourne, C.1.
January 30, 1946.

THE INTERVERTEBRAL DISK.

SIR: In 1931 the Medical Research Council of Great Britain published their Special Report No. 161 with the title: "The Intervertebral Disks: Observations on their Normal and Morbid Anatomy in Relation to Certain Spinal Deformities." The observations were made by Dr. O. A. Beadle, Radcliff Travelling Fellow, who worked under Dr. Schmorl in Dresden.

Dr. Schmorl started in 1925 to remove every spine for examination as a post-mortem routine. In five years he had removed and examined 7,000 spines. His museum contained 600 complete spines and many hundred smaller preparations of parts of spines. Hundreds more spines were ready for mounting in the museum when the necessary space was available.

As it is not permissible in English-speaking countries to remove the whole spine from the body, it is very unlikely that any more exhaustive and finished survey of the intervertebral disk will be made for many years to come. It is also very probable that the events of the past six years have caused a wide dispersion of Dr. Schmorl's unique collection.

Many articles on the role of the intervertebral disk in producing low back pain have been written during the past few years. To the best of my knowledge in only one instance—an article by A. C. Mooney in the *British Journal of Radiology* of May, 1945—has Beadle's monumental monograph been mentioned.

It is felt that were the existence of Beadle's monograph more generally known, many of the problems connected with lesions of the disks could be more readily clarified, and statements such as that made by one of your contributors in the journal of January 12, 1946, that there was a likelihood of regeneration of the *nucleus pulposus* unless the cavity was freely curetted would not be made.

Yours, etc.,

W. H. GODBY.

179B, St. George's Terrace,
Perth,
February 4, 1946.

SIR: At a meeting of the New South Wales Branch of the British Medical Association held on July 26, 1945, Dr. Douglas Miller is reported as having said (THE MEDICAL JOURNAL OF AUSTRALIA, January 12, 1946, page 58): "Localization by observation of pain, numbness and of objective signs referred to a dermatome might be deceptive." In his letter of August 31 (THE MEDICAL JOURNAL OF AUSTRALIA, September 15, 1945, page 351) Dr. Miller stated: "No one can doubt the good effects of conservative surgery on the intractable sciatica . . . where localization can be accurate . . ."

Would Dr. Miller please inform me what happened during this period of 36 days to make him change his opinion or is his use of the word "accurate" not so accurate?

Yours, etc.,

MARION A. RADCLIFFE-TAYLOR.

Perth,
Western Australia,
January 30, 1946.

THE SURGICAL AND APPLIED ANATOMY OF THE INGUINAL REGION.

SIR: Lieutenant-Colonel Harley Turnbull and others interested who may not have had access to recent literature may be interested in references to the anatomy and surgery of the inguinal regions.

The references to the former concern the accurate anatomy of the inguinal region based on painstaking dissection by

Barry J. Anson and Chester McVay. The references are: Barry J. Anson and Chester B. McVay: "Inguinal Hernia. I. The Anatomy of the Region", *Surgery, Gynecology and Obstetrics*, Volume LXVI, February, 1938, page 186; "The Anatomy of the Inguinal and Hypogastric Regions of the Abdominal Wall", *Anatomical Record*, Volume LXX, January, 1938, page 211.

In regard to the surgery of hernia, there is an excellent article by Lieutenant-Colonel W. J. M. Brandon to be found in *The Lancet*, February 10, 1945, page 167. Lieutenant-Colonel Brandon has pointed out that the frequency of recurrence of oblique inguinal hernia is due to one factor alone: "That the true internal ring is over-lapped completely by the lower border of the internal oblique muscle. In consequence, closure of the so-called ring at operation does not occur at the true internal ring, but at a point lower down the cord."

Those who are interested should read the article themselves. One comment concerning the operative treatment by Lieutenant-Colonel Brandon is worth noting, namely: "In this way almost every conceivable type of complication may be dealt with, and almost every conceivable mood of the surgeon satisfied; and at the same time a sufficiently shock-proof method of primary repair can be effected without involving the patient, and particularly the soldier, in yards of *fascia lata* or binding him with fetters of floss silk, and one that may yet save the merchant seaman from becoming a sort of animated ironclad."

Two other useful references to the treatment of direct inguinal hernia are: Leo M. Zimmerman: "Clinical Surgery: Inguinal Hernia: II. The Surgical Treatment of Direct Inguinal Hernia", *Surgery, Gynecology and Obstetrics*, Volume LXVI, February, 1938, page 193. This is based on the work of Anson and McVay. And a second reference on the use of heterogenous fascial grafts is: Jacob Chandy: "The Use of Heterogenous Fascial Grafts in the Radical Operation for Hernia", *Annals of Surgery*, Volume CXXII, November, 1945, page 885.

In conclusion may I draw attention to a very nice piece of applied anatomy in regard to sliding hernia: Roswell K. Brown: "Sliding or Paraperitoneal Hernia of the Pelvic Colon", *Surgery, Gynecology and Obstetrics*, Volume LXXVI, January, 1943, page 91.

Yours, etc.,

E. S. MEYERS.

The University of Queensland Medical School,
Brisbane,
February 5, 1946.

Obituary.

ANDREW ERIC ASPINALL AND ARCHIBALD JOHN ASPINALL.

THE late Reverend A. Ashworth Aspinall, for many years principal of the Scots College, Sydney, had five children, four sons and one daughter, all of whom graduated in medicine at the University of Sydney. One of the sons, Robert, lost his life in the war of 1914-1918 while he was serving with the Australian Army Medical Corps. The other three sons served in the same war, and the eldest, Archibald John, always known as Archie, served also in the war that has just ended. Andrew Eric and Archibald John died recently of the same kind of cardiac condition within a few weeks of one another, the former on November 10, 1945, and the latter on December 5, 1945.

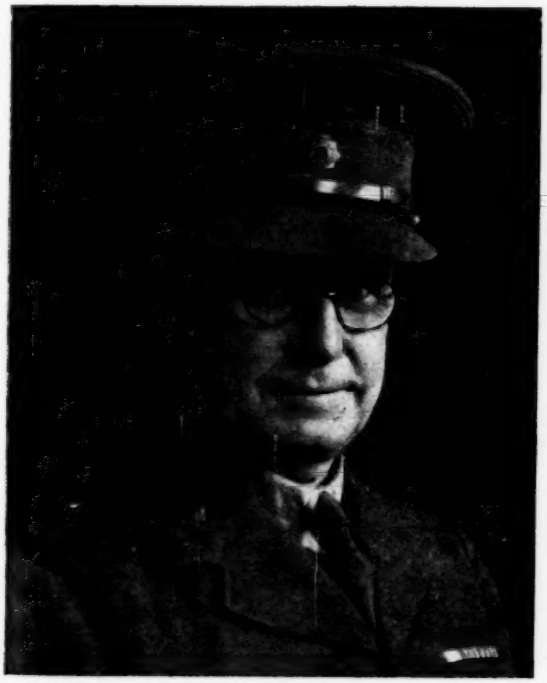
ANDREW ERIC ASPINALL graduated in medicine at the University of Sydney in 1915. He had been a student at Sydney Hospital and became a resident medical officer on graduation. He soon went on active service, however, as his three brothers did. He served as regimental medical officer and later as anæsthetist to a surgical team. He was promoted to the rank of major. On his return to civil life, he married and settled in practice at Turramurra. In 1923 he was elected to the staff of the Royal North Shore Hospital of Sydney on the medical side. At the time of his death he was physician to indoor patients. He took an interest in school education and was medical officer to Knox College. In his undergraduate days he played football and later in life became a devotee of golf. Nine years before his death, however, he had a coronary occlusion and was advised to give up work. This he would not do, and for seven years after his illness he conducted a busy practice. The strain told on him and he eventually had to rest. For twelve months before his death he had been able to do no work at all; shortly before his death he was feeling well. He is survived by his wife and three daughters.

Dr. E. P. Blashki writes: Dr. Andrew Eric Aspinall, who passed away at his home at Turramurra on November 10, was the third son of the late Reverend A. Ashworth Aspinall. He was educated at the Scots College and graduated at Sydney in March, 1915. In the first Great War he enlisted in 1915, served in Egypt and France as regimental medical officer of the Fourth Divisional Engineers and transferred to the Second Australian Casualty Clearing Station in 1917, serving until demobilized. On his return in 1919 he commenced practice at Turramurra. His inclination was towards clinical medicine, and he held the post of assistant physician and later physician at the Royal North Shore Hospital until 1944, when he was compelled by ill health to retire, and was then appointed to the consultant staff.

Eric Aspinall had an extensive practice; his patients appreciated greatly his sound knowledge of medicine, his unfailing kindness and his devotion to duty. He was a good all-round athlete. At the university he was captain of the second football team. As a student he always had the confidence and respect of his fellows, who repeatedly elected him their representative. He enjoyed a like measure of popularity among his colleagues on the hospital staff and elsewhere. He is survived by his widow, three daughters, of whom the eldest is married to Captain W. Inglis, Australian Army Medical Corps, and the second to Flight Lieutenant George Blaxland, and one grandson.

ARCHIBALD JOHN ASPINALL was the eldest of the sons of A. Ashworth Aspinall. Like the other members of the family, he graduated in medicine at the University of Sydney, taking his degrees in 1906. He took part in much of the life of the university, being "representative" of his year and playing games. He was a member of the "second fifteen" of the university and he played football and cricket for Saint Andrew's College. On graduation in 1906 he became resident medical officer at Sydney Hospital and began an association which was broken only by his death. His second year at the hospital was spent as resident radiologist and pathologist, and during his third year he was "first senior". In this office he had the task of instructing resident medical officers in anæsthesia, and he did this with great success. Then followed three years in the office of medical superintendent. He had organizing ability which was recognized by both the honorary and resident staffs. He was able to see the broad aspects of a problem and also its implications; with this he had the gift of being able to pay attention to detail. This was shown in his surgical work and was one of the reasons for his success. It was during Aspinall's tenure of the office of superintendent that the clinical school was established at Sydney Hospital. In earlier years medical students of the University of Sydney had done all their clinical work at the Prince Alfred Hospital—the institution had not then acquired the title "Royal". Aspinall's share in the inauguration of the clinical school was considerable. In due course he went into private practice and was appointed honorary assistant surgeon; this was in 1912. In 1914 war broke out; Aspinall was a member of the Army Medical Corps, and he left Australia with the First Field

Ambulance. This was an interesting unit. B. J. Newmarch was in command and held the rank of lieutenant-colonel; R. H. Millard and E. S. Stokes were majors; the captains were J. B. St. Vincent Welch, A. J. Aspinall, L. W. Dunlop, H. R. G. Poate, C. E. Wassell and W. E. Kay. Of these, only Dunlop and Poate are living. The ambulance was present at the landing on Gallipoli. Aspinall and Poate were detailed to remain on the transport, which at once began to receive wounded. With J. B. Newmarch these two officers engaged in what was really a ferry service for wounded. They had twenty men from the ambulance and no equipment other than ambulance panniers. With more than 600 obviously wounded men on board at a time and a journey of about three and a half days to Alexandria, both ingenuity and endurance were taxed to the uttermost. About twelve men died every day and were buried at sea, Aspinall and Poate taking it in turns to read the burial service. After journeying for four months between Gallipoli, Alexandria and Malta, Aspinall and his companions rejoined the ambulance on Gallipoli. Unfortunately Aspinall became ill and had to leave before the evacuation. He was sent home



to Australia in the latter end of 1916. The second world war found Aspinall anxious to serve once more. Two useful parts were assigned to him. First he was liaison medical officer on the Dutch hospital ship *Oranje* and later he became commanding officer of the 114th Australian General Hospital at Goulburn.

In civil life after the war of 1914-1918 Aspinall devoted himself with characteristic energy to his surgical work. In 1923 he joined the full staff of Sydney Hospital as honorary surgeon. On his retirement recently he was senior honorary surgeon and he was appointed to the consultant staff. In 1931 he was appointed lecturer in clinical surgery. He held appointments at other institutions. He was senior honorary surgeon of the Eastern Suburbs Hospital, Sydney, honorary consulting surgeon at the Royal South Sydney Hospital, honorary surgeon at the Women's Hospital, Sydney, and consulting surgeon to the Australian Jockey Club. He was a foundation Fellow of the Royal Australasian College of Surgeons and a member of its State Committee for New South Wales. He was a member of the board of the Scottish Hospital.

Outside the sphere of medicine Aspinall was interested in general education. He was a member of the Council of Saint Andrew's College within the University of Sydney; he also served on the councils of the Scots College, the Presbyterian Ladies' College, Croydon, and the Presbyterian Ladies' College, Pymble.

To set out Aspinall's appointments and to name the institutions and bodies with which he was associated show that he lived a full life. He was a methodical and careful worker; he was ingenious and often showed his ingenuity in the devising of splints for the treatment of patients with unusual injuries. He was a man of high ideals and transparent honesty and he had a gift of friendship. Men trusted him and were not disappointed. He did not like his name, Archibald, but preferred to be known, and always was known, as Archie. If he had to be reported as taking part in a medical meeting he preferred to be mentioned as "Archie" rather than as "Archibald" or "A.J.". On one occasion in his early years at Sydney Hospital this had a surprising result. A patient from the out-door department wrote to him and addressed the envelope to Dr. Ah Chee Aspinall. His joy and delight seemed to know no bounds. As a matter of fact no account of him would be complete without reference to his lively sense of humour and his gift of mimicry. The serious and important "honoraries" who brought their complaints and sometimes their importance to his superintendent's office, had their affairs attended to, but they also tickled his sense of humour and added to his store of anecdotes. When he was in the mood he was without an equal as a Sydney Hospital *raconteur*.

As a final word it may be said of Archie Aspinall that as a surgeon and a man he had qualities that set him apart as one worthy of emulation. He has left a lasting impression on the "body and soul" of the hospital that he served with such long and consistent devotion.

Dr. J. G. Edwards writes: So Archibald John Aspinall has passed on. Known to the profession as "Archie", he was dear to all who were privileged to know him in his student, hospital and later professional days.

He was a lovable character, upright and honest in all his ways, and his first thought was always of others and never of self. As a resident medical officer at Sydney Hospital and later as medical superintendent he showed rare kindness and great ability as a surgeon and administrator. To patients he devoted all his energy, and no effort was too great to ensure the welfare of those under his care.

During his many years as assistant and honorary surgeon he carried out his duties most efficiently, and there has probably never been a member of that staff with sounder surgical judgement, especially in acute abdominal cases. He was also in the front rank of teachers and had the knack of imparting knowledge in such a manner that it remained forever in one's memory. He had a wonderful "tissue sense", never using unnecessary violence; like his teacher, MacCormick, he "caressed" the tissues, and as a consequence his patients enjoyed a rapid convalescence.

He served with distinction in the first Great War and sacrificed a growing practice to serve his country. Although over age, he again offered his services in this war, but was rejected on medical grounds. Still determined to serve his country, he gained an appointment as liaison medical officer in the hospital ship *Oranje*, where he served for eighteen months. For his services he received a high decoration from the Netherlands Government. Later he organized the 114th Australian General Hospital at Goulburn, and once again his organizing ability placed this institution in the front line of hospital units. Ill health brought him back to consultant practice, but he was not able to work to full capacity.

He was a delightful companion at any gathering; his sense of humour was keen but kindly, his power of mimicry delightful, and he possessed a fund of anecdotes about early days at Sydney Hospital. His memory is pleasant to all of us who knew him and his life will always be an example of duty well done. All his old friends will feel the deepest regret that he will no longer be able to enliven their remaining years with his cheerful and bright presence.

Professor W. Keith Inglis writes: The death of Archie Aspinall has removed one who will be sorely missed by his colleagues and friends. His was a wide interest for good in many fields of endeavour, which extended beyond the practice of medicine.

I can speak in terms of deep appreciation of the interest he took in the scientific side of medicine. He and the late Dr. Ralph Worrall were representatives of the honorary medical staff on the Board of Directors of the Sydney Hospital soon after the end of the first world war, and it was largely the result of their efforts that the Kanematsu Institute of Pathology became established at the Sydney Hospital.

It was in no small measure due to Archie Aspinall that the *esprit de corps* in this institution was maintained at such

a high level. His loyalty to his predecessors and colleagues on the honorary staff of Sydney Hospital was unbounded; he not only respected them, he loved them. He knew all their little idiosyncrasies, and, as he was a good mimic, his stories of early days at the hospital were greatly appreciated.

Others will speak of his concern for the welfare of students, of his untiring efforts on behalf of nurses, of his setting aside of personal interests to give of his best in the fighting services during both world wars, but, for those who knew him well, the feeling at present is that of deep personal loss in the passing of one who was a wise counsellor and a staunch friend, to whom one never went in vain for guidance and sympathetic understanding.

GEORGE McLEAN.

We regret to announce the death of Dr. George McLean, which occurred on February 6, 1946, at Chatswood, New South Wales.

Naval, Military and Air Force.

THE TRAINING PRIOR TO DISCHARGE OF CERTAIN SERVICE MEDICAL OFFICERS.

The following notice is published at the request of the Director-General of Medical Services of the Royal Australian Air Force.

Further to the notice concerning the above scheme which appeared in the journal of January 5, 1946, it is desired to bring to the attention of former medical officers of the Royal Australian Air Force details of the procedures to be followed in making application for an approved course of refresher training under the terms of the scheme, or for claiming ninety days' pay and allowances if an approved course has already been completed.

Application should be made direct to the Director-General of Medical Services, Royal Australian Air Force. On receipt of the application, the Director-General of Medical Services will forward to the applicant copies of the appropriate *pro forma*. This should be completed in duplicate and returned to the Director-General of Medical Services accompanied by the statutory declaration and statements as indicated on the *pro forma*.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Evans, Cyril Percival Victorious, M.B., B.S., 1943 (Univ. Sydney), 8, Andrew Street, West Ryde.

Bracken, Derek Christopher Gordon, M.B., B.S., 1940 (Univ. Sydney), 81, Darling Point Road, Edgecliff.

McManis, Arthur Geoffrey, M.B., B.S., 1936 (Univ. Sydney), 283, Cleveland Street, Redfern.

Ireland, Joseph Frank, M.B., B.S., 1940 (Univ. Sydney), NX104569, Captain, 1, Transvaal Avenue, Double Bay.

Scott-Young, Norman Reginald, M.B., B.S., 1941 (Univ. Sydney), 108, Wanganella Street, Balgowlah.

Moseley, Moira Shirley, M.B., B.S., 1943 (Univ. Sydney), 94A, Birriga Road, Bellevue Hill.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Weber, Leighton Otto Davis, M.B., 1943, B.S., 1944 (Univ. Sydney), 522, Melbourne Road, Frankston.

The undermentioned has applied for election as a member of the Tasmanian Branch of the British Medical Association:

Parkinson, Arthur, M.B., B.S., 1925 (Univ. Melbourne), Queenstown, Tasmania.

Books Received.

"Principles of Human Physiology" (originally "Starling's Principles of Human Physiology"), by C. Lovatt Evans, D.Sc., F.R.C.P., F.R.S., LL.D. (Birmingham); with section on the special senses by H. Hartridge, M.A., M.D., Sc.D., F.R.S.; Ninth Edition; 1945. London: J. and A. Churchill, Limited. 9¼" × 6", pp. 1165, with many illustrations. Price: 36s.

"Chest Examination: The Correlation of Physical and X-Ray Findings in Diseases of the Lung", by Richard R. Trail, M.C., M.A., M.D. (Aberdeen), F.R.C.P. (London), with a Foreword by Sir Walter L. Langdon-Brown, M.A., M.D., F.R.C.P., D.Sc., LL.D.; Second Edition; 1495. London: J. and A. Churchill Limited. 8½" × 5½", pp. 135, with 94 illustrations. Price: 12s. 6d.

Diary for the Monty.

FEB. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 21.—South Australian Branch, B.M.A.: Council Meeting.
FEB. 21.—Victorian Branch, B.M.A.: Executive Meeting.
FEB. 22.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 26.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 27.—Victorian Branch, B.M.A.: Council Meeting.
FEB. 28.—South Australian Branch, B.M.A.: Scientific Meeting.
MARCH 1.—Queensland Branch, B.M.A.: Branch Meeting.
MARCH 2.—Tasmanian Branch, B.M.A.: Annual Meeting.
MARCH 5.—Federal Council, B.M.A., in Australia: Meeting at Sydney.
MARCH 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MARCH 6.—Western Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All Public Health Department appointments.

Editorial Notices.

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